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THE DIAGNOSIS, TREATMENT AND IMMEDIATE PROGNOSIS OF CEREBRAL TRAUMA*

An Introductory Study of 1494 Cases

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ON April 27, 1933 the *New England Journal of Medicine*[1] published an editorial which stated that "the diagnosis, prognosis and treatment, therefore, of cranial injury after head trauma are in an unsettled state." (From the context it is evident that "cerebral" not "cranial injury" is meant.) Further the editorial goes on to say that "the more modern devices of treatment, such as the use of intravenous hypertonic solutions, repeated lumbar puncture, and even subtemporal decompression of the brain are all methods not as yet proved to be either essential or even valuable in the treatment of this condition." Taken by itself such a sweeping condemnation of the present methods of treatment of brain injuries amply justifies critical examination. In addition it should not be forgotten that "cranial (cerebral) injury after head trauma" is no longer a problem reserved for solution by the specialist, whether neurologist or neurosurgeon. Thanks to the automobile every practising physician is having these cases deposited daily, as it were, on his very doorstep, and in the main has learned to care for them very efficiently, partly through personal experience, and partly through the perusal of the mass of literature that has collected on this subject in the past fifteen years. This experience has taught him that lumbar puncture judiciously used may be a life-saving measure, that hypertonic solutions given intravenously are very often a prompt and efficient method of meeting a grave surgical emergency and that subtemporal decompression used as a panacea in the treatment of "fractured skulls" as advocated in the decade from 1910 to 1920, and even as recently as 1933[2] kills very much more frequently than it cures. If this therapeusis by lumbar puncture and dehydration is ineffective, and if the diagnosis and prognosis of this condition rest on such a shaky foundation, the physician should be so informed. If this situation does not exist, however, that fact should be made apparent also for this is no academic question. The correct answer affects the lives and well-being of many hundreds of people, and the most efficient treatment should be a matter of common knowledge.

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The quoted editorial is based upon a paper by W. R. Russell recently published in *Brain*[3]. Although much of the content is undeniably correct, the paper as a whole is unconvincing, principally because it is based on a series of only 200 cases, from which moreover all the cases dying within twenty-four hours of injury have been extracted. On such data the author makes assertions regarding the diagnosis and treatment of acute brain injuries that are admittedly at variance with the well-substantiated opinions of experienced surgeons. Russell states: (1) that "Common contre-coup injury is caused by the brain tearing itself from its covering by the force of its own momentum" (p. 577); (2) that "increase of intracranial pressure provides a powerful means of controlling hemorrhage from torn cerebral capillaries and veins" (p. 597); and (3) that "Morphia is useful—and can apparently be given without danger" (p. 598). Such unsound physiopathologic concepts involving as they do the cause, the progress and the treatment of these injuries can best be discussed under the appropriate headings below.

In this and succeeding papers I propose to present data in regard to diagnosis, immediate prognosis and treatment of craniocerebral injury drawn from a four year experience with 1494 patients at the Boston City Hospital. This should demonstrate that the diagnosis and treatment of this condition are well understood, that the immediate prognosis is reasonably good and that the modern devices of treatment are both essential and valuable.

SUMMARY OF MATERIAL. Most of the large series of cases of craniocerebral injury have been studied from a postmortem point of view. Clinical studies have been inaccurate and inconclusive because, having in mind the multiplicity of symptoms that result, the number of cases studied has been too few. This paper deals with certain aspects of 1494 cases seen and studied by the entire surgical staff of the Boston City Hospital over a period of four and one-half years. These cases fall into two groups. The first group are those cared for on the general surgical service. The second group were treated on the Neurosurgical Service and were under the direct care of the author. (Chart 1.) The general surgical material consists of 989 cases with a mortality of 17.6 per cent, but

a mortality of 5.7 per cent if deaths within twenty-four hours are omitted. The rest of the material has an excessive per cent of operated patients. This is because there is a natural gravitation of the more seriously injured toward the special Neurosurgical Service and away from the general surgical side. There are 505 of these cases with a mortality of 15.6 per cent and a mortality, without "twenty-four hour deaths", of 9.9 per cent. From this combined material it has been possible to get significant data in regard to the diagnosis and treatment of compound and depressed fractures, extra- and subdural hemorrhages and other less common types of brain injury. This is being reserved for later publication. The figures presented below have to do only with the common types of cerebral injury: concussion, congestion and edema, contusion, and laceration. All cases are excluded that left the hospital against the surgeon's advice. In the majority of the fatalities the cause of death was verified through the kindness of the medical examiner's office.

CHART 1

	Total Cases	Total Deaths	Mortality %	Deaths After 24 • Period	Mortality % Without 24 • Deaths
General Surgical Service	989	175	17.6%	50	5.7%
Neuro-Surgical Service	505	79	15.6%	47	9.9%
Boston City Hospital	1494	254	17.0%	97	7.2%

CHART 2

- **Skull**
  - Linear and/or Comminuted Fracture
  - Depressed Fracture
  - Compound Fracture
  - Special Fractures
    - Involving Para-Nasal Sinuses
    - Middle and/or External Ear
    - Including Pneumoencephalocele
    - Cribriform Plate
    - Bullet Wounds
- **Brain**
  - Concussion
  - Edema
  - Contusion and/or Laceration
  - Special
    - Hemorrhage
      - Sub or Extra Dural
      - Rarer Forms
        - Sub-pial
        - Sub-cortical
        - Intra-ventricular
        - Rupture of Venous Sinus
        - Multiple Petechial
    - Infection
      - Meningitis
      - Cortical Abscess
- **Scalp**
  - Lacerated and/or Incised Wound
  - Contusion
  - Hematoma
- **General Bodily Conditions**
  - Surgical Shock
  - Exhaustion
  - Dehydration

DIAGNOSIS. *General Bodily Conditions:* Accuracy of diagnosis, based on as firm a pathological foundation as possible, followed by appropriate treatment is the road to success in the treatment of any surgical condition. This first requirement is met by the classification of these skull and brain injuries into groups in accord-

ance with chart 2. Of the general bodily conditions I wish particularly to emphasize again the importance of being positive about the presence or absence of surgical shock. This diagnosis is based on a falling pulse pressure, a low temperature, a pale moist skin and a weak rapid pulse. It is essential to recognize this condition because all examinations are contraindicated while it is present and treatment must be limited to measures suitable for the shock alone, regardless of any other evident pathology.

Skull: So far as injury limited to the skull is concerned, compound fracture is diagnosed on entrance to the hospital by palpation through the scalp wound, this method having proved infinitely more accurate than x-ray examination. The diagnosis of other fractures (with one exception) by x-ray or other means is postponed until after convalescence from the brain injury has begun, i.e., during the second or third week. At that time views in the antero-posterior and postero-anterior positions as well as stereoscopic lateral films from both sides are taken. The single exception is when the presence of an extradural hemorrhage is in question. Here the positive visualization of a linear fracture crossing some part of one or both of the middle meningeal arteries may turn the tables in favor of operative interference and is information that is essential. I am convinced that x-ray examinations made except as above are not only useless in a large percentage of cases but are indeed definitely harmful to the patient.

Brain Injuries: Classification of the brain injuries is more complicated and because of this the surgeon must always hold himself ready at any time to revise or completely change his working diagnosis and therefore his treatment. A proper understanding of the chronology of the pathology is essential. Following a blow upon the head strong enough to jar the brain, unconsciousness may be produced. This primary unconsciousness is due to *concussion,* a term that should be scrupulously reserved for this preliminary symptom only. G. Gavin Miller[10] in 1927 produced true concussion experimentally and in addition repeated and corroborated previous experimental work by others. He demonstrates that concussion is an entity by itself, that it is caused only by mechanical means (i.e., a blow on the head), and that it is immediate in onset and tends of itself to spontaneous recovery without sequelae unless severe enough to paralyze respiration. The condition should be sharply differentiated from all brain injuries showing gross lesions such as contusion, multiple petechial hemorrhages, edema and the like. At present concussion is best explained as due to a direct mechanical derangement of the molecules within the nerve cells, causing temporary loss of function. Such a disturbance is quite a different matter from gross cerebral lesions, although in both cases, if his theory be accepted, the damage is done by the same type of trauma, the molecular field being involved in concussion and the microscopic or macroscopic fields in the other types of injury. Cases suffering only from concussion rarely reach the hospital, indeed I have been able to recognize concussion among hospitalized patients only in young children who have been admitted for observation as a precautionary measure. Objectively and subjectively these patients are normal and have a normal intracranial pressure and cerebrospinal fluid and require no active treatment. If the blow be harder and the injury more severe, there will be a more prolonged period of deeper unconsciousness alternating with periods of delirious behavior and succeeded in its turn by absolute flaccid coma, respiratory difficulty, collapse of the circulation and death from respiratory paralysis. This group of symptoms follows immediately after, and may merge with, the unconsciousness due to the concussion. They are caused by gross brain damage, the severity of which, other things being equal, is, in general, in direct relation to the length and depth of the coma. This damage can be divided into three recognizable groups, (1) edema and congestion, (2) contusion and (3) laceration. Each succeeding group includes as part of its pathology the previous preceding groups, as for example edema and contusion are both present in association with a laceration of the brain. They overlap and often the classification must be arbitrary, but at operation or autopsy one or the other of these pathological features will predominate. Any of the conditions classed under "rare forms" may be superadded to any one of the fundamental groups. A lacerated contused brain may be in addition subjected to pressure from a subdural hematoma providing a cortical vessel is torn and bleeds into the proper space. Edema and congestion of the brain will be associated with multiple petechial hemorrhages and the same condition may form the background for the changes brought about by the development of an extradural hemorrhage.

Excepting in pure concussion, the primary reaction of the brain to injury is like that of soft tissues anywhere else in the body. There is congestion, edema and a rise in the pressure of the cerebral venous circulation. This is accompanied by a concomitant rise in intracranial pressure[11, 12]. If this process is continued long enough to produce anoxemia there is tissue damage and more edema[7, 13]. Clinically it is possible to recognize this *congestion and edema.* The patient has been unconscious, complains of a headache, may have a short period of memory loss, in children convulsions and even hemiplegia may be present and altered tendon and abnormal reflexes occur. The intracranial pressure is moderately raised in the majority of cases and the cerebrospinal fluid is clear and

colorless and of normal chemical content. The brain volume is increased but there is no mechanical obstruction to the absorption of cerebrospinal fluid.

In *contusion* the unconsciousness is more prolonged as is the amnesia; the headache more severe; there may be disorientation and mild confusion; and abnormal reflexes are common. There may be a period when surgical shock is present right after the injury. The cerebrospinal fluid is pink or pinkish yellow with increased protein content if there has been any hemolysis of red blood cells. The intracranial pressure is moderately elevated and tends to remain up for two to five days in spite of repeated lumbar drainage. There is not only increased brain volume but also an acute mechanical hydrocephalus. This is due to the blocking of the avenues of absorption in the arachnoid villi by free red blood cells until such times as they are removed by the action of the meningocytes[14].

In *laceration* a state of surgical shock is practically always present; irrationality, confusion and disorientation may be extreme or there may be deep coma with general muscular flaccidity including the sphincters. Respiratory periodicity, a slowed pulse and high pulse pressure, a rise in temperature, stiffness of the neck, fixed dilated pupils (a late sign), a slow return of consciousness with severe headaches and a prolonged amnesic period are usual signs and symptoms. Reflex changes are common and signs of excessive local cortical damage such as aphasia, astereognosis, facial palsy and hemiparesis occur frequently. The intracranial pressure is very high (I have measured as high as 100 mm. mercury) and repeated lumbar drainage over a week to ten days will usually be necessary to reduce it to normal levels. The cerebrospinal fluid is at first red or appears almost like pure blood, changing later to dirty yellow and then clear yellow, this latter color often remaining after the increased intracranial pressure has again become fixed within normal limits. The chemical content is as under contusion. There is increased brain volume of only a moderate degree together with a marked mechanical acute hydrocephalus as in contusion. This if untreated may lead later to a mild permanent distention of the ventricles.

The diagnosis of the *extra* and *subdural hemorrhages, meningitis* and the rarer types of brain injury must be deferred for later presentation but it can be stated here that their symptoms and signs are superimposed upon those of the fundamental conditions epitomized above and cannot be intelligently evaluated until that fact is appreciated.

Much of this cerebral damage is done by *contre-coup injury*. Attention was directed to this fact by Russell[3] as noted above. His explanation of the mechanism however is incorrect and cannot be allowed to stand without comment. There can no longer be any question but that the skull in life is practically a closed box and that the small amount of motion allowed the brain inside this box is due to the to-and-fro movement of the cerebrospinal fluid which in turn is possible only through the so-called elastic doors (occipito-atlantoid ligaments, etc.[5]). LeCount and Apfelbach[8] and B. M. Vance[9] have demonstrated that contre-coup lesions occur only when the patient's head is injured while in motion. In such circumstances the brain lags behind and is most closely applied to the aftercoming portion of the skull. To be sure the skull is flattened at the point of impact and the brain immediately beneath is directly injured by this local application of force. In addition, however, because the axis of the skull is abruptly shortened at right angles to the plane of impact and because the brain is most closely applied to the bone directly opposite the point of impact, the most severe bruising occurs at this place. They further point out that contre-coup subdural hematomata from torn cortical vessels are one of the most common accompaniments of fractures and that the same lesion due to tearing loose of the tributaries of the large sinuses is relatively rare, a relationship that would not exist if the brain were free to move to any significant extent as postulated by Russell.

TREATMENT. In the past and even in the present[2] the only choice in treatment has been considered by many to lie between the watchful waiting of rest in bed and the opposite extreme of operative interference, usually in the form of a subtemporal decompression. More recently, however, thanks to the work of Weed[12], Foley and Putnam[15] and others with hypertonic and hypotonic solutions intravenously, and saturated solutions by the bowel, in association with a freer use of lumbar drainage checked by manometric readings (Frazier[16], Landon[17], Jackson[18], Fremont-Smith[19], Ayer[20], Munro[21]) the pendulum has gradually swung away from these earlier methods. In the rush of enthusiasm attending the adoption of new methods many instances of misapplication have cropped up and, as a result, there is a tendency at present to blame the failures on the method, rather than on the operator for his improper use of it.

Therapeutic Dehydration: All that any hypertonic solution intravenously, or a saturated solution of magnesium sulphate by the bowel, can do or should be expected to do in this connection, is to reduce the brain volume. The intravenous method will do this more rapidly and for a shorter period of time than the use of the salt by the bowel. Coincidentally if the intracranial pressure be high, it will be reduced by just so much as the brain is shrunk, but on

the other hand no change can or should be expected to take place in the residual increased intracranial pressure that is present because of the acute hydrocephalus brought about by the mechanical blockade of the absorptive channels. Therefore universal treatment of all forms of brain injury by this one type of therapy cannot be justified. Furthermore dehydration may be dangerous if used without accurate measure on its effect. I have unintentionally produced toxicity (with symptoms closely simulating those of acute edema or contusion) both by the use of 50 per cent glucose intravenously and by saturated solution of magnesium sulphate given by rectum. Also those who have used hypertonic salt solution know to their cost what a devastating effect the secondary wave of cerebral edema that follows the therapeutic brain shrinkage may have. When properly limited, however, to cases of edema (where it can be the sole therapeutic agent) or to use as a preliminary emergency treatment in cases of contusion and laceration, both 50 per cent glucose intravenously (repeated once or twice if necessary) and saturated solution of magnesium sulphate by rectum (repeated every three or four hours for four or three doses) will prove of very real value and will often be actually a life-saving measure. The effect of both of these drugs should be checked by manometer readings of the cerebrospinal fluid pressure.

Lumbar Puncture: Decompression by lumbar puncture, on the other hand, is suitable for all three types of uncomplicated brain injuries. The edema is relieved because with the reduction of intracranial pressure to normal, the venous congestion is corrected and the reactivated circulation allows the partially damaged asphyxiated cells to recover as much as possible. The excess intracellular and perivascular fluid is then absorbed and the brain volume is thus returned to normal. In contusions and lacerations the excess unabsorbed fluid is mechanically removed together with a small amount of free blood, and the meningocytes, if the drainage be repeated often enough, are aided in the uncorking of the absorptive channels in the arachnoidal villi[14, 23]. Here, too, venous congestion and intracranial hypertension are reduced just as in the edema group. Rest in bed, like ice to the head, or a quiet dark room, proper nursing, etc., is important but as compared with these other more direct methods, can do little to correct the pathology.

Subtemporal Decompression: Subtemporal decompression by its very nature is only efficient in the mild cases in which it is not urgently needed. In the severe cases the decompressive effect is shortly lost because the dural and bony opening is soon tightly plugged by an edematous cortex. This is as effective a cork as though the dura had been left unopened. Enlarging the decompressive opening beyond the limits of the temporal muscle seldom proves efficient, causes great difficulty at the closure and adds future trouble in the form of cortical adhesions.

Arrest of Intracranial Hemorrhage: The most pernicious of all teachings relative to the treatment of craniocerebral injury is the misconception that an increase in intracranial pressure will prevent or arrest intracranial hemorrhage associated with such an injury. This erroneous point of view can be most clearly stated by quoting in full from Russell's paper[3]: "It is important to bear in mind that a degree of increase of intracranial pressure provides a powerful means of controlling hemorrhage from torn capillaries and veins. In this way cerebral edema may prevent hemorrhage and correspondingly the artificial reduction of intracranial pressure may aggravate it." That this assertion is grossly inaccurate and not in accord with the facts has been shown experimentally and is known clinically to all surgeons with any wide degree of experience in traumatic or any other form of intracranial surgery. This fallacy is widespread among the profession and should be done away with as rapidly and as completely as possible.

Experimentally in 1928, H. S. Forbes[4], working first alone and later in the same year with H. G. Wolff[5], confirmed the findings that Cushing[6] published in 1901 and "add further details of a quantitative nature". They demonstrate that following a rise in intracranial pressure there is a slowing of the blood flow in and dilatation of the veins and arteries, the circulation being maintained (without increase in the systemic blood pressure) by a rise in pressure in the cerebral capillaries, arterioles and smaller arteries. Following a further rise in intracranial pressure the cerebral circulation becomes even slower and then begins to fail; as a result of bulbar anemia the systemic arterial pressure then rises reflexly, and the cerebral circulation is reëstablished. This compensation may occur several times if the intracranial pressure is raised by steps. Such slowing of the capillary circulation (congestion) is equivalent to anoxemia applied to the tissues of the brain generally. Depending upon the degree and the length of time it is present, there is a proportional cellular destruction, petechial softening, edema, further stasis and stagnation, and capillary hemorrhage. Small areas of destruction may coalesce into larger areas with further spreading edema and further rise in intracranial pressure. Such a succession of events even in the absence of injury has been reported by Cobb and Hubbard[7] as a series of cerebral hemorrhages from venous and capillary stasis.

Clinically it is common practice for the surgeon to open a skull and dura in the presence of

increased intracranial pressure and find a congested bleeding cortical vein in the operative field. The surrounding cortex will be bulging, not pulsating and firmly jammed against the edges of the bony opening. All attempts to close the ruptured vein and stop the hemorrhage fail until the bony and dural openings are enlarged to a point where decompression is accomplished, and high intracranial pressure thus reduced to normal. As soon as this is done the cortex no longer bulges, pulsation is again resumed, the cortical veins shrink and lose their congestion, and bleeding from the ruptured vein in question can be easily controlled by a silver clip, a silk tie or even the implantation of a muscle "stamp" graft; the vein if small enough may even close itself.

These observations show that cerebral venous and capillary pressures are approximately equal to intracranial (cerebrospinal fluid) pressure, and that they rise and fall with it. Thus high intracranial pressure cannot cause hemostasis without causing anoxemia and necrosis of the brain. By the same token lowering a high intracranial pressure by lumbar puncture decreases venous bleeding, because the venous pressure falls with the lowering of the cerebrospinal fluid pressure. The venous pressure is high *because* the intracranial pressure is high. Arterial pressure is rarely a factor since it is not affected until the intracranial and cerebral venous pressures approach arterial diastolic pressure, i.e., 900 to 1100 mm. of water. Obviously arterial bleeding can never be checked by increased intracranial pressure; capillary stasis and death would take place long before such an intracranial pressure (1600 to 2000 mm. of water) could act on the arterial stream[22].

The Use of Morphia: Associated with the foregoing in its danger to the patient but more widespread in its effect because of the immediate results obtained is the use of morphia in craniocerebral injuries. Any statement that "morphia is useful and can apparently be given without danger", is most pernicious teaching and cannot be condemned too strongly. Morphia is a dangerous drug and one of its chief actions is to depress respiration. Of these injuries concussion alone often alters and even paralyzes the respiratory center in the medulla. Increased intracranial pressure usually impairs the function of this same mechanism. Since death occurs in many cerebral injuries from respiratory paralysis, it is folly to add the burden of morphin poisoning to an already embarrassed vital respiratory centre. This is not mere theory. Any physician called as consultant to see patients with brain injury has again and again acted as mere spectator to a respiratory death hastened by the injudicious use of morphin. It can be taken as a good general rule: "No morphine where increased intracranial pressure is suspected."

Procedures: The fundamental treatment of the 505 neurosurgical cases included herewith has been a combination of therapeutic dehydration and decompression by lumbar drainage. In those cases that are not immediately fatal this treatment is expected to cause a progressive improvement in the patient's signs and symptoms. If this does not occur, and toxic dehydration and meningitis can be ruled out, exploratory temporal trephine is considered indicated. This serves to eliminate the possibility of sub- or extradural hemorrhage or of one of the rarer forms of brain injury. It can be categorically stated that in no case in this series has the medical examiner made a diagnosis of death due to lumbar puncture.

Value of Therapeutic Lumbar Puncture: For the purpose of comparison of mortality rates between this and the more passive forms of treatment I have grouped the cases from the general surgical services into those punctured and those not punctured. (Chart 3.) The difference in mortality is quite striking and speaks for itself. These cases were treated by house-officers under the direction of the surgical staff and cover a period of four and a half years.

CHART 3

	Total Cases	Deaths	Mortality %
Treatment by Lumbar Puncture	340	32	9.4%
Treatment without Lumbar Puncture	631	122	19.3%
Total Cases	989	175	17.6%

CHART 4

Service	Total Cases	Total Deaths	Mortality %	Cases Treated by Lumbar Puncture %
1st				
1922	65	16	24.6%	23%
1930-33	224	34	15.1%	22%
2nd				
1922	62	17	27.4%	16.1%
1930-33	170	22	12.9%	57.0%
3rd				
1922	94	14	14.8%	68.7%
1930-33	124	24	19.3%	30.0%
4th				
1922	97	14	18.1%	11.6%
1930-33	85	18	21.1%	35.2%
5th				
1930-33	80	11	13.7%	44.8%

This same group when arranged by services and separated into cases treated in 1922 when lumbar puncture was first introduced and those treated in the past three years shows less striking but similar figures. (Chart 4.) In particu-

lar it is interesting to note the drop in the second service mortality from 27 to 13 per cent while there is an associated rise in the percentage of cases treated by lumbar puncture from 16 to 57 per cent. Conversely on the third service a fall in the percentage of lumbar puncture treated cases from 68 to 30 per cent has been accompanied by a rise in service mortality of from 15 to 19 per cent. The apparently contradictory figures under the fourth service are not representative on account of the small number of cases involved. These facts and the figures tabulated under prognosis demonstrate that the intelligent use of therapeutic dehydration as a supplement to decompression by repeated lumbar puncture drainage is the safest and best method of treating the large majority of brain injuries. Wholesale condemnation of this method must be due either to a lack of experience or a failure to grasp the possibilities and limitations of the therapeusis.

PROGNOSIS. While it is possibly true that most brain injury cases that will not survive the first twenty-four hour period without treatment may not survive with treatment, this statement cannot be taken so literally as to imply that intelligent oversight by a surgeon during this time will not save a few lives that would otherwise be lost. The fatalistic attitude recently publicly taken by Dandy[2] that "20 per cent (of the total number of patients with severe injuries to the head) must be regarded as beyond redemption by any rational means available" and that another 10 per cent can only be saved by a subtemporal decompression, pushes the surgical clock back ten years. Such claims can only be explained by the supposition that the clinic from which he writes, but whose figures he does not quote, must have a relatively small number of these cases. (Chart 5.)

and hence to institute adequate treatment. Only the unavoidable pulmonary and meningeal infections are considered as excusable cause for such deaths. Among those cases on whom I have learned to make an early and fairly accurate diagnosis the mortality drops further. For example: If the unusual middle

CHART 6

NEURO-SURGICAL SERIES

	Living	Dead	Mortality %
Total Cases	505	79	15.6%
Total Cases without Meningeal Hemorrhages	465	61	13.1%
Total Cases without Meningeal Hemorrhages and Compound Fractures	405	42	10.3%

meningeal and subdural hemorrhages are eliminated from this series, the rate is only 13.1 per cent. If a further subtraction of the compound fracture cases as being a problem of sepsis rather than brain injury is made, the figure again declines, reaching a minimum of 10.3 per cent. It is evident then, that in the present state of our knowledge, the proper use of therapeutic dehydration and lumbar puncture decompression applied in accordance with a diagnosis based upon a proper conception of the pathology of brain injury, should permit only about 10 per cent of patients suffering from the ordinary forms of skull and brain injury to die. If all possible types of skull and brain injuries are included the mortality should be not over 16 per cent. The greatest opportunity for improvement lies in an earlier and more accurate diagnosis of the meningeal hemorrhages, and certain rarer forms of brain injury, and an increasingly earnest attempt to elimi-

CHART 5

	Totals		Living		Dead		% Mortality	
	Neuro-Surgical	Rest of Hospital	Neuro-Surgical	Rest of Hospital	Neuro-Surgical	Rest of Hospital	Neuro-Surgical	Rest of Hospital
Concussion*	0	496	0	492	0	4		0.8%
Edema, Contusion and Laceration	376	339	335	243	41	96	10.9%	28.0%
Rupt. of Middle Meningeal Artery	10	7	3	1	7	6	70.0%	85.0%
Subdural Hematoma	30	33	19	1	11	32	36.6%	90.6%
Compound Fracture	60	47	41	19	19	28	31.6%	59.5%
Depressed Fracture	29	23	28	14	1	9	3.4%	39.1%
Period Covered	3½ yrs.	4 yrs.						

*Under "Rest of Hospital" this diagnosis "Concussion" includes all forms of brain injury as well as linear fractures of the skull. Under "Neuro-Surgical" the diagnosis of "Concussion" is used only as described in the text.

The mortality in my own group of 505 patients is 15.6 per cent. (Chart 6.) Furthermore it has been my experience that any case of skull or brain injury that dies after the first thirty-six hours, providing they have been brought promptly to the hospital, does so because of a failure on my part to make a correct diagnosis

nate postoperative sepsis in compound fractures of the skull.

SUMMARY AND CONCLUSIONS

1. The diagnosis of craniocerebral injury is firmly established on a pathological basis. It includes linear, depressed and compound frac-

tures; concussion, edema, contusion and laceration of the brain, extra- and subdural hemorrhage, and a few other less common forms of skull and brain injury.

2. The immediate prognosis of cerebral injury after head trauma can be fairly good. In this series of 1494 cases the mortality was 17 per cent. In a group of 405 cases of the common types of brain and skull injury, the mortality was 10.3 per cent. A 20 per cent group mortality implies inaccurate diagnosis, improperly applied treatment or lack of experience with a sufficiently large number of cases.

3. One treatment for the usual forms of brain injury has been standardized. It consists of the preliminary treatment of surgical shock, when present, followed by therapeutic dehydration and repeated lumbar puncture for decompression. Treatment of compound and depressed fractures, meningeal hemorrhage, and the less common forms of brain injury is not considered in this paper.

4. If improvement does not set in and continue under this treatment, and if toxic dehydration and meningitis can be ruled out, bilateral exploratory temporal trephines are indicated to eliminate the possibility of undiagnosed sub- or extradural hemorrhage, or one of the less common types of brain injury.

5. Cases treated as above, or by lumbar puncture alone, have a mortality nine per cent less than similar cases treated without lumbar puncture.

6. In over 1000 lumbar punctures for decompression in acute brain injuries there has been no death attributable to this procedure.

7. Therapeutic dehydration is suitable treatment for traumatic edema of the brain. In combination with decompression by lumbar puncture it is suitable treatment for traumatic contusion and laceration of the brain. Improperly used it may cause unexpected and severe symptoms.

8. The use of morphin is dangerous in cases where increased intracranial pressure is suspected. It should never be administered under such circumstances.

9. Increased intracranial pressure does not aid in the prevention of bleeding from the cerebral vessels.

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AMEBIASIS IN CONNECTICUT*

BY JOHN H. FOSTER, M.D.†

REPORTS of amebic dysentery among visitors to the World's Fair in Chicago during the past few months have called the attention of the whole country as well as of the medical profession to the importance of this disease. New England probably has received its proportion of acute and chronic amebic infection from this new national focus of infection. Two cases of amebic dysentery were discovered in Waterbury as soon as the notice was sent out from Chicago.

Amebiasis is still considered a tropical disease, but there have been many warnings from public health authorities over the high incidence of carriers throughout the United States as dis-

*Read before the Waterbury Medical Association, December 14, 1933.

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covered by the routine surveys by Kofoid[1], Boeck and Stiles[2], Craig[3], Johnstone, David and Reed[4] and others. In 1932, 100 cases of amebic dysentery were reported to the United States Public Health Service[5] from California and nearly 500 cases from Louisiana. It is not a reportable disease in most states so no accounts are available from the whole country. It is probably much more common in the northern states than is generally realized. It has seemed to me timely to call attention to amebic infection as seen in Connecticut and to note some of the forms the disease may take.

In 1923 Blake and Hiscock[6] from the New Haven Hospital reported nine cases occurring in a small epidemic in Ansonia, Conn. They had seven cases of amebic dysentery and two of liver abscess. (The source of that epidemic is still uncertain and only one more case was discovered subsequently.)

In the ten years since that report up to the time of the Chicago incident, there have been twenty cases of amebic infection with fifteen deaths reported to the State Department of Health of Connecticut[5]. In addition there have been eight cases in the last two months, six of them originating in Chicago.

During the last four years I have seen eight cases of amebic infection in my practice which have been of sufficient interest to report more or less in detail, as they illustrate some of the types of infection encountered and emphasize the importance of recognition of amebic infection even in Connecticut.

CASE 1, A. S. C. (1246), a 33-year-old male, was seen in 1930 complaining of diarrhea, abdominal distress, gas and lower abdominal pain of four weeks' duration. In his past history Mr. C. had lived six years in different parts of China as a teacher and in the consular service. He had an attack of dysentery in Indo-China on his return to the United States in 1921 which lasted six months. Ever since this he has had a tendency to loose stools but an examination at Johns Hopkins Hospital in 1926 was entirely negative.

Stool examinations showed mucus and cysts of amebae. Sigmoidoscopic examination showed superficial ulcerations. Treatment was given of colonic irrigations, emetine, yatren and stovarsol for a period of six weeks and cleared up the symptoms which have not returned.

CASE 2, Mrs. L., 42 years of age (2237), came to see me in February, 1933, complaining of pain in the left upper abdomen, general weakness, palpitation and gas of several months' duration. She gave a history of having lived in Shanghai for several years up to 1928 but never having had dysentery. Feces examination showed cysts resembling entameba histolytica, and sigmoidoscopic inspection revealed a low grade of colitis. On treatment with rectal instillation of 1:100 solution of quinine and with stovarsol by mouth her symptoms cleared up, she gained weight and has been much improved. The pain in the region of her splenic flexure has disappeared completely.

An example of the chronic ulcerative colitis was CASE 3, Mr. W. G. (2006), 52 years of age, who complained of attacks of colitis off and on from 1914, at which time he lived in Ansonia. Seen in May, 1932, he was having frequent stools with mucus and at times blood. His general health was poor, he was underweight, weak, had palpitation and tightness in his chest when climbing hills and some nocturia.

Mr. G. weighed 130 lbs., was very pale and almost cachectic in appearance. Sigmoidoscopic examination showed a chronically ulcerated sigmoid and rectum with membranous patches. Many motile amebae containing RBC were seen in the fresh smears. He responded immediately to stovarsol therapy and gained 30 lbs. in two months. He is still free from symptoms and he claims to be in perfect health, although I have not examined his colon since.

A case with more acute symptoms was seen in a patient from Naugatuck (1018), CASE 4, in July, 1933. I was attending the man's wife at the time who was suffering from a chronic mucus colitis which was definitely not of amebic origin. Mr. W., 44 years of age, while in Cleveland in June, was taken with an attack of diarrhea and for three to four weeks had been having frequent stools especially at night, not loose, but the feces surrounded with fresh mucus and blood. Stools showed typical vegetative amebae and sigmoidoscopic examination showed ulcerative and membranous colitis. Scrapings from the membrane showed the motile amebae containing many RBC. A course of stovarsol caused his symptoms to clear up so promptly that I could not get him to come in for a check-up for nearly four months, during which time he was symptom-free. Mr. W. had been at the Congress Hotel in Chicago three or four weeks before the onset of his dysentery.

In October, 1933, I had the privilege of seeing a patient, Mr. A. W. (CASE 5), 49 years of age, in consultation with Dr. S. J. Mullins in Danbury.

This man had attended the Chicago Fair in September. About ten days after his return he felt ill, and developed pain in his lower abdomen. Dr. Mullins found a temperature of 100°, tenderness in the right lower quadrant, a leucocyte count of 12,000, 84% polys, and diagnosed an acute appendicitis. The patient refused operation, but symptoms increased and two days later and after a consultation an appendectomy was performed. A swollen and definitely suppurative retrocecal appendix was found and removed.

He was making a good recovery when about ten days after the operation he began to pass small amounts of blood and mucus. The stools increased in frequency so that he was having from 20-30 stools daily. His temperature was 102-103° and pulse from 120-140. He was failing rapidly and was in a critical condition when amebae were demonstrated in his stools. Sigmoidoscopic examination showed a very severely ulcerated rectum and sigmoid with a very foul odor to the discharge. Typical motile RBC containing amebae were present in the mucus scraped from the membrane and ulcers. Emetine, 1 gr. daily intramuscularly, carbarsone 0.25 grams twice daily were started. He improved greatly within a few days and has made a good recovery although his convalescence has been slow.

Pathological report on the appendix by Dr. J. O. Collins of the Waterbury Hospital:

Sections (figs. 1 to 4) show an appendix which has been split on one side. The lumen of the organ contains a moderate amount of exudate, desquamated epithelium and cellular débris. Within the exudate are noted a number of spherical bodies about 35 microns in diameter which stain a bluish pink with hematoxylin and eosin. Some of these spherical bodies contain small vacuoles and red

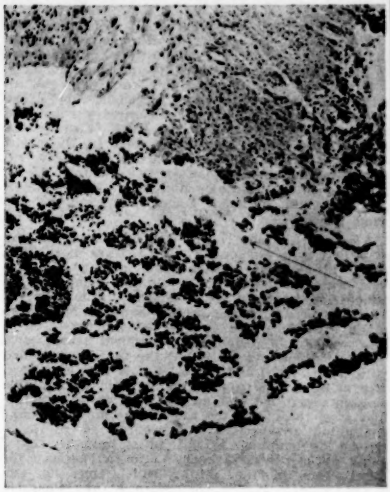

FIG. 1. Low power (× 100)* view of appendix with ameba in the pus in the lumen.

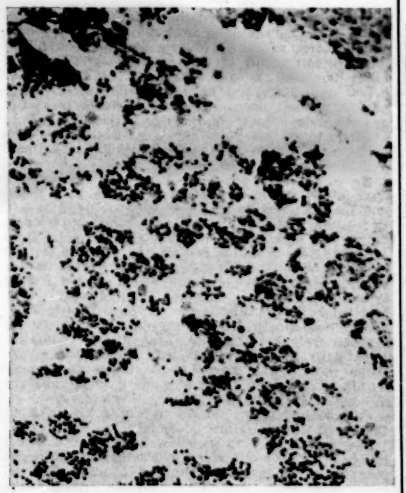

FIG. 2. Higher power (× 375)* of same section showing ameba more distinctly.

*Editor's Note: This illustration was slightly reduced.

FIG. 3. Section of mucosa and submucosa of appendix showing larger number of entameba histolytica.

FIG. 4. Higher power of same section showing ameba in more detail containing vacuoles and red cells.

Vol. 2
193

blood cells; their size and morphology are consistent with a diagnosis of amebae. The mucosa is interrupted in a number of places by ulceration. Amebae are found in large numbers in the floor of these ulcers. The ulcers are quite deep, some of them extending into the submucosa. The mucosa is congested and shows scattered areas of hemorrhage. Polymorphonuclear leukocytes and eosinophilic leukocytes are rather numerous in the mucosa. The submucosa and muscle layers show definite infiltration with all types of wandering cells among which polynuclear leukocytes are predominant. There is one small area of suppuration in the muscle layer. There is rather marked fibroblastic activity throughout the wall of the organ. The serosa is decidedly thickened by proliferating fibrous tissue in which are numerous infiltrating round cells and plasma cells.

Diagnosis: Amebic ulcers of appendiceal mucosa with subacute appendicitis.

Sub Note*.

CASE 6, Mr. A. J., an Italian, 47 years of age, gave a history of severe attacks of ulcerative colitis in 1925 and 1928 each lasting for several months, which cleared up after prolonged treatment with rest, diet and irrigations, bismuth and foreign protein therapy. Amebae were never demonstrated in his stools. For the past five years he has been free from symptoms although not enjoying really good health. In November of this year he had a recurrence of his diarrhea with some blood and mucus. Questionable amebae were found in a stool examination but definite RBC containing amebae were found in the mucus obtained from a sigmoidoscopic examination. The mucus membrane showed irregular areas of inflamed mucosa, hyperemic but not ulcerated. Treatment has been emetine and carbarsone with prompt improvement in all his symptoms.

The following case, M. H. (CASE 7), was first seen in 1922 in Changsha, China, at the age of two and has been seen at intervals since then. At that time she seemed fairly normal except for a tendency toward anemia and lack of energy. In June of that year she left with her parents on a furlough traveling almost continuously for the next months. Just before arriving in the United States in October, she began to have a bloody diarrhea. She was in the New Haven Hospital under the care of Dr. Grover Powers with few interruptions from November, 1923 to August, 1924. Her symptoms were chiefly diarrhea and a fever which at times reached 102°. Amebae were reported in the first stool examination, but could not be confirmed. She was given emetine on the basis of the clinical picture but with no apparent improvement. She continued to have a low-grade fever and diarrhea. There was a moderate secondary anemia, white counts that varied between 20,000 and 30,000 and, occasionally, small amounts of blood in the stool, but repeated stool examinations were negative.

The last day of February the child was readmitted with a temperature of 103°, and she remained in the hospital this time for five months. She was seen by all the prominent medical people in New Haven and by a number of visiting pediatricians both American and European. Stool examinations to the number of about 200 were made, urine examinations, x-rays, cultures of blood, urine and feces, precipitin tests of urine, animal inoculations, Wassermann and agglutination tests, all were negative. The secondary anemia increased and the white blood count was consistently high, reaching 47,000 in March. The spleen became slightly enlarged and there were a few enlarged venules on the abdominal wall but no ascites. An exploratory laparotomy revealed a large indurated liver with increase in the connective tissue lying between the lobules and some points of apparently central, fatty degeneration. A lymph node removed at the time of operation was negative on microscopic examination. In addition to emetine,—santonin, neoarsphenamine and tonsillectomy, were tried, all with very little effect on either the bloody diarrhea, the fever, or the anemia. The fever gradually became less and a series of five transfusions were begun at the end of May; 150 cc. of citrated whole blood was given each time with apparently beneficial effect. When she was discharged from the observation of the clinic, she was about normal weight and height, had 4,280,000 red cells per c.mm.; her temperature still was slightly elevated, (99°) and there was diarrhea and the stools contained blood. The diagnosis with which she returned to China was "idiopathic tropical cirrhosis of the liver". Further transfusions were recommended and a diet free from roughage. Prognosis was poor, as that little understood condition was generally regarded as fatal.

During the autumn in Changsha her condition remained about the same. The red cells remained over 4,000,000 and, in spite of occasional exacerbations of the diarrhea, she seemed quite well. Many stool examinations were made but it was not until February 24th, about sixteen months after the onset of the disease, that amebae were undoubtedly found in the stool. Emetine was given again, at first in quarter grain doses, later the doses were increased to one grain but only nine grains were given in five weeks' time. A great deal has been written of the resistance of cysts to emetine, the so-called emetine fast cases, so yatren (chiniofon) was next used. She later had a relapse with a temperature 100-101°. Her blood count dropped to 3,800,000. She was given an intraperitoneal transfusion of 300 cc. of citrated blood. The improvement from this time on was marked. She seemed more vigorous. The liver returned to normal size. The blood disappeared from the stool and the consistency became firmer and more normal. The control of defecation which had become very poor did not return to the usual state until early in 1926. She had abdominal pain during the next year, but in general her health was much improved. Stovarsol was given for four courses during the year. Since 1927 she has lived in New England. From March, 1928 to June, 1931 she had recurring attacks of bloody diarrhea with fever of 103-104°, and with stools positive for amebae. She has been in the hospital in Hanover, New Hampshire, and in the Children's Hospital in Boston for observation and study. Treatment has consisted of emetine, bismuth, neoarsphenamine, bismarsen and gentian violet which were given at various times up to March 1933, although no amebae have been found since July, 1931. Her present condition seems to be satisfactory.

CASE 8 is a nurse, 38 years of age, now living in Danbury. From 1921 to 1925 she was in China. In June, 1922 she had an acute enterocolitis and a low-grade appendicitis. Later she had an appendectomy but was not well all summer. In October she had acute catarrhal jaundice but was well from then on to the winter of 1924-1925, when she developed bronchitis and ran a persistent low fever of 99-100°. She had vague abdominal pain but no colitis. There were suspicious signs in her chest and by x-rays, and as she had a family history of tuberculosis she was returned to the United States in

*Surgeons resent the criticisms in some of the newspaper and medical reports of cases of amebiasis being operated upon for appendicitis. From the pathological findings in this case it would seem that the diagnosis of acute appendicitis was correctly made and that appendectomy was definitely indicated.

the summer of 1925 and was sent to the Gaylord Farm Sanatorium, Wallingford, Conn., for four months' observation.

After careful study she was discharged with the diagnosis undetermined, but they decided she did not have tuberculosis. Dr. A. B. Dayton in New Haven after saline purging found amebae and small ulcers in the sigmoid. Her symptoms promptly improved with emetine treatment. She still has occasional recurrences of abdominal distress and fever.

THE ENTAMEBA HISTOLYTICA

The entameba histolytica is the only human pathogenic ameba. It enters the body through the mouth in the encysted form and is in itself evidence of fecal contamination from some preceding case of amebiasis. This protozoan makes its habitat in the mucosa of the colon where through its proteolytic power it buries itself in the submucosa and lives and reproduces indefinitely. No symptoms may be noted for months or years. The entameba histolytica is held in check by the body resistance, the vegetative form remaining in the mucosa. The amebae escaping in the lumen become contracted and are excreted in the feces in the encysted form. This is the latent or carrier state.

Exacerbations of activity due to various causes produce increased ulcerations of the intestinal mucosa, an ulcerative colitis, with typical symptoms of dysentery. With the increased peristalsis the mucus contains blood and numbers of amebae in the motile or vegetative stage.

From the usual location in the submucosa of the colon the amebae may be carried to other organs and there have been many cases reported of this so-called systemic type of amebiasis[7]. As illustrations abscesses of lung, liver, brain, spleen, hepatitis and ulcers of the skin may be cited. Amebae have been reported in the urine, semen and in cases of iritis, thyroiditis and salpingitis.

Kofoid reported finding amebae in the lymph glands of Hodgkin's disease and in the bone marrow in cases of arthritis deformans. His views, however, have never been very widely accepted. Boeck[8] has recently shown that bone involvement is very unlikely and that Hodgkin's disease is not associated with amebic infections. He points out, however, that small amebic lymphatic ulcers may act as definite foci of bacterial systemic infections.

It is important to remember that it is only in the encysted stage that the ameba can be transmitted to a new host. The vegetative forms which are present in the acute dysenteric stage die very soon after exposure to air. It is even difficult to keep them alive long enough for satisfactory laboratory examination.

It might be well also to recall the experiments of Walker and Sellards[9] in 1914 in the Philippines who fed amebic cysts to 20 volunteers in a Manila prison. Of the 20, two (10%) never did show any cysts. Of the 18 who showed cysts and became carriers only 4 (20%) developed symptoms of dysentery and they came down at periods of 20, 57, 87, and 98 days after the feeding experiment. None of the 20 men fed with vegetative amebae developed dysentery or became carriers.

Diagnosis: The most important point is to consider the possibility of its presence. Amebae should be suspected in all cases of colitis and the stools should be carefully examined microscopically. The technique of stool examination is described in many textbooks but is often confusing. Training and experience are necessary.

In the acute stage the motile ameba should be present and usually in large numbers in the mucus. They can be seen with the low power lens but under the high power the characteristic irregular shape, the ameboid movements and appearance of RBC inclusions verify the diagnosis. They are usually 30-50 m. in diameter. The warm stage and stool are not absolutely necessary, but the stool should be fresh. There are certain characteristics in the microscopic picture in the fresh smear of a stool from an amebic dysentery as opposed to bacillary dysentery that help in differentiation. The amebic stool is quite free from bacteria, the mucus will contain many RBC but comparatively few WBC. Charco-Lyden crystals are often present and there is not much fecal débris. The bacillary stools contain many bacteria and pus cells with a smaller number of RBC and much detritus. The long-standing chronic amebic ulcerative colitis may be complicated with secondary pyogenic infection which will give the appearance of the bacillary stool but ameba should also be present.

It is the diagnosis of the cystic stage that offers most difficulty and requires the trained protozoölogist. Even they insist on smears stained with hematoxylin and iron for identification of the cysts. The technique is difficult and the diagnosis is uncertain and controversial. My own method is to ascertain the presence of cysts by making a fresh stool smear in 1:000 eosine solution, in which the cysts appear as clear refractile bodies, counterstaining with Lugol's solution, which brings out the nuclei. If amebic cysts are present the therapeutic test with emetine or stovarsol is applied. The difficulty in diagnosis in the systemic type is illustrated in case 7. There literally hundreds of stool specimens were examined before amebae finally appeared. In cases of amebic abscess it is often impossible to demonstrate the ameba. Stool cultures can be used in doubtful cases.

EPIDEMIOLOGY

In cases 1, 2, 7 and 8 the source of infection was in China. Surveys in Peking by Kessell and Faust showed that about 35 per cent of the

native Chinese were carriers of entameba histolytic cysts and about 25 per cent of the foreigners. Eight to ten per cent is reported to be the incidence in the United States.

Case 3 apparently carried his infection for eighteen to twenty years from a probable infection in Ansonia. There was no apparent transmission to other members of his family.

The etiology in cases 4 and 6 was puzzling until the announcement from the Chicago Board of Health.

TREATMENT

There are several drugs that have a specific amebicidal power. First used was ipecac, and with its active principle emetine they were for years the most effective preparation known, but toxic effects often appeared before cure was obtained. Yatren (or chiniofon) is less toxic but has not proved reliable. Acetarsone (or stovarsol) is more effective but considerably more toxic due to its high arsenic content. Carbarsone[10] is a recent amebicide recommended and seems to hold much promise. It is an arsenical (28% arsenic content, 4 carbamino-phenol-arsenic acid) but it is much less toxic than stovarsol and has a therapeutic index eight times greater. It also has the advantage of being considerably less expensive. Vioform[11] is an oxyquinoline preparation belonging to the same group as yatren which has been highly recommended, but with which I have had no personal experience.

COMMENT

The number of cases noted would hardly justify their presentation but for the fact that all of these individuals had symptoms which had resisted treatment, some of them for years and from many physicians, but which responded to specific amebicidal therapy when the etiological agent was discovered.

It is well to call attention again to the symptoms other than dysenteric, viz., the low grade gastro-intestinal complaints in cases 2 and 6, the hepatitis in case 7, also the acute appendicitis in case five which was of amebic origin.

No studies have been made of the incidence of amebic cyst infection in the contacts of the persons studied but no cases with symptoms of amebiasis were found in any of their households.

SUMMARY

(1) There have been over 30 cases of amebiasis reported to the State Department of Health in Connecticut in the last ten years with 15 deaths.

(2) Details of eight cases are given illustrating various types of acute and chronic infection. Four of the cases originated in China, two contracted their infection in Chicago. The source of infection in the other two cases is not clear.

(3) The symptoms varied considerably, vague gastro-intestinal symptoms in two, diarrhea, blood and mucus in the stools in others with varying degrees of prostration. Pyrexia and hepatitis in one and an acute suppurative amebic appendicitis in another.

(4) The therapy of amebiasis is highly specific. Of the various amebicides available, emetine, yatren, acetarsone, vioform and carbarsone, the last-named seems to be the safest and most effective.

(5) Amebiasis is already endemic in Connecticut and there will probably be more cases developing as a result of the Chicago epidemic. Amebic infection should always be considered in the diagnosis of gastro-intestinal disorders.

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PAPILLARY CARCINOMA OF THE RENAL PELVIS

Report of a Case

BY H. A. CHAMBERLIN, M.D.,* AND H. E. MAC MAHON, M.D.*

TUMORS arising from the transitional epithelium of the urinary tract are found most commonly in the bladder, and only rarely in the pelvis of the kidney. In both of these sites they usually assume one of three rather characteristic forms, namely, simple papilloma, papillary carcinoma, and invasive tumors resembling the squamous cell carcinomas of the skin. A papillary carcinoma of the renal pelvis is a comparatively rare tumor, and cases diagnosed correctly on the basis of careful clinical and roentgenological studies before operation warrant reporting. The number of cases in the literature is still small and due in part to this, the diagnosis is often extremely difficult.

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REVIEW OF THE LITERATURE

Very recently MacKenzie and Ratner[1] reported a case in which a transitional-cell papilloma of the pelvis of the kidney showed evidence of malignant transformation. They reviewed the literature bearing on tumors of the renal pelvis, mentioning the early papers of Albarran[2], Albarran and Imbert[3], Hunt[4], Thomson-Walker[5], and Meltzer[6]. In their own series of ninety-six cases of tumor arising within the kidney, only one was a primary growth of the renal pelvis. In textbooks of pathology, little or no space is given to this group of tumors. Sirter[7] in describing the papillomata of the kidney pelvis, pointed out the resemblance of these tumors to papillomata of the bladder, and showed that they may appear as multiple tumors of the pelvis, ureter and bladder. Sirter believed that cases of bleeding from one kidney showing papillomata of the bladder, especially if these are situated about the ureteral orifice, should be considered as examples of multiple tumors, with similar papillary lesions in the renal pelvis.

CASE REPORT

Clinical History:

G. H. B.—White male, 71 years of age, was admitted to the Cambridge Hospital Oct. 16, 1932. About three months before entry he passed on several occasions, dark muddy urine. At that time, there was no frequency, dysuria, or abdominal or lumbar pain. About one month later he noticed that his urine was again discolored, a condition which recurred about twice a week during the succeeding two months. During this time he felt intermittent pains, often severe, in the left lumbar region, and lost both strength and weight. For the past two weeks he has passed no clear urine.

Physical Examination:

A well-developed, and fairly well-nourished, feeble elderly male, rational, but dull and listless. An abdominal examination was negative with the exception of slight tenderness on firm pressure over the left kidney. Neither kidney was palpable. The external genitalia and prostate were essentially normal.

Cystoscopic Examination (October 17):

The bladder mucosa was normal except for slight congestion over the trigone. There was beginning trabeculation, but no intravesical enlargement of the prostate. The right ureter was catheterized with ease, and normal urine was obtained. On the left side, the catheter passed readily to the junction of the pelvis and ureter, but could not be passed into the pelvis, and no urine could be obtained.

Roentgenologic Examination of the Kidneys:

Pyelograms were made of both kidneys. They were rather obscured by gas, but appeared of about regular size and shape. The right kidney was easily filled with the injected fluid, (12% sodium iodide solution) and was quite normal. The left showed a very poor and incomplete filling (see fig. 1). The impression from a study of the pyelogram on the left side was that there was a pathological lesion within the pelvis, either a tumor or blood clot, which prevented a normal emptying, and at the same time prevented the catheter from entering the pelvis.

The Laboratory Findings:

The urine was brown, of neutral reaction with sp. g. 1.034. Sugar was absent. There was a trace of albumen, and an abundant sediment showing many red blood cells, and ten to fifteen white blood cells per high power field. The phenolsulphonphthalein test was unsatisfactory because of the discolorization, due to the presence of blood. The blood non-protein nitrogen was elevated to 66 mg. per 100 cc. A blood count showed 4,100,000 R. B. C., 11,600 W. B. C., and Hbg. 80 per cent. The differential white count was 84 per cent polymorphonuclear leucocytes, 14 per cent lymphocytes, and 2 per cent mononuclear cells. The platelets were not diminished, and the R. B. C. showed no changes in size, shape, or staining.

FIG. NO. 1. Photograph of pyelogram showing normal right kidney pelvis, and filling defect of the left. The arrow indicates the location of the papillary tumor, and just above this the dark, round area represents a dilated calyx filled with sodium iodide solution.

Course of Illness:

For three days following the cystoscopic examination, the patient's condition remained unchanged. On the fourth day, the temperature rose to 101°F., and the patient complained of moderately severe pain in the left upper quadrant and left lumbar region, with point of greatest tenderness and spasm situated over the left kidney. Several days later, a definite mass was palpated in this area. The temperature and pulse remained elevated, pain and tenderness increased, and the patient was very ill, and without question was losing ground rapidly.

On October 23, one week after his admission, the left kidney was operated upon under local novocaine anaesthesia. It was greatly enlarged, distended, and surrounded by very firm dense inflammatory adhesions. A nephrostomy was performed, allowing a large quantity of turbid urine to drain from the kidney pelvis, and a drainage tube was inserted into the renal pelvis. It did not seem advisable at this time, because of the patient's poor condition, to thoroughly explore the kidney. The patient, during the succeeding days, improved steadily, and on the fifth day following this operation, the temperature was again normal.

On November 14, three weeks after the nephrostomy, a nephrectomy was performed under spinal anaesthesia. The kidney, the pelvis, and the upper part of the ureter were surrounded by very firm dense inflammatory tissue, so that the pelvis could not be satisfactorily palpated. Because of the difficulty in reducing the pedicle to a size suitable for ligation, and a desire to finish the operation as soon as possible, a large clamp was left on the pedicle without attempting to ligate it. The kidney on section showed a large papillary tumor arising from the pelvic wall which will be described in detail below.

The convalescence was uneventful, the clamp was loosened on the fifth and removed on the sixth day after the nephrectomy. The patient was discharged on November 29, two weeks after this second operation. At this time the blood non-protein nitrogen had dropped to 26 mg. per 100 cc. and the phenolsulphonphthalein output was within normal limits.

In March, 1933, four months after leaving the hospital, the patient was again examined. His general health had steadily improved, he had gained ten pounds, and with the exception of slight and transient dizziness felt very well. A roentgen examination of the lungs and all bones was made at this time, but no evidence of metastasis could be demonstrated. An abdominal examination was essentially negative. A left ureterogram showed no filling defect in the remaining portion of the ureter. The bladder mucosa appeared quite normal.

Now, on September 15, 1933, nearly a year since the operation, the patient's daughter reports that her father is in excellent health, and that he has gained an additional ten pounds since the date of the last examination.

Pathological Report (S-32-480)

The kidney from this patient showed many features of unusual interest. The tumor lying in the pelvis had caused a damming back of urine which later had become infected. This led to a chronic destructive pyelitis, a pyelonephritis, and an infection of the perirenal fat tissue.

Gross Description of Specimen:

The specimen consists of the left kidney together with considerable surrounding fat tissue. The perirenal fat and connective tissue are unusually firm and are riddled with small petechial hemorrhages, and small areas of necrosis and cellular infiltration (fig. 2). The true connective tissue capsule strips very easily from a rather uneven kidney surface. Yellow rosette-like areas 1-2 mm. in diameter and bordered by a fine red zone are scattered irregularly over the surface of the kidney. Section of the kidney shows narrow yellow lines, radiating up from the pelvis throughout both cortex and medulla. The parenchyma is pale, swollen, and rather edematous. The renal arteries and their larger branches have thickened walls. The pelvis and calices are dilated and contain coagulated blood, some turbid fluid, and soft, irregular masses of gray, glistening, partly necrotic tissue. The surface of the pelvis has a blood-stained reddish-gray, thickened, opaque appearance. The papillae are here and there flattened out, forming a base to the dilated triangular calyx. The veins at the margin of the cortex and medulla are slightly injected so that these two portions of the kidney are clearly differentiated from one another.

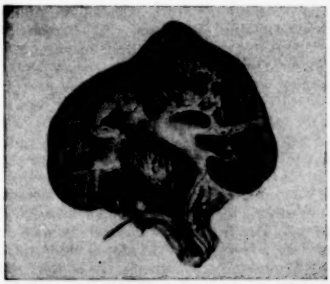

FIG. NO. 2. The papillary tumor lies in the lower portion of the kidney pelvis. The narrow stalk is clearly visible showing how the tumor could serve as a ball-valve to the uretero-pelvic opening.

Just at the margin of the pelvis and ureter is a tumor growth, approximately the size of a small prune, measuring 3.5 x 3.5 x 3.5 cm. This is growing as a papillary nodule which is attached by a single narrow glistening pedicle. The pedicle itself is quite soft and there is no induration at the attachment of this pedicle to the pelvis of the kidney. The surface of the tumor mass is blood-stained, soft, and gray. At some points it is rough and ragged and extremely friable, and it is quite obvious that portions from the periphery of the tumor have desquamated into the pelvis of the kidney. Here and there over the surface of the tumor and at different points along the surface of the pelvis there are small calcified concretions. The tumor growth is hanging down into the dilated conical upper end of the ureter. In this position it has acted as a partial obstruction to the outflow of urine leading to hydronephrosis and its complications.

Microscopic Description:

Several sections were taken from the kidney itself and from different portions of the tumor, the stalk of the tumor and the attachment of the stalk to the pelvis. The tumor shows an unusually varied picture. There are places in which it resembles the classical slowly growing benign papilloma of the bladder with a delicate connective tissue stroma

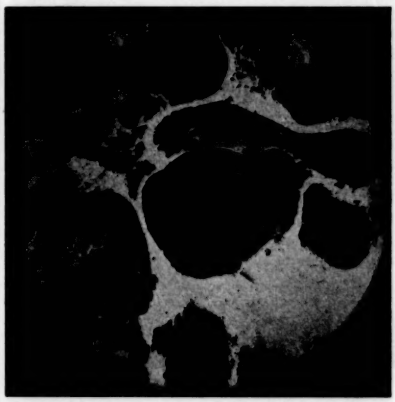

FIG. NO. 3. Section of the tumor showing villous-like processes. There is a delicate connective tissue stroma rich in capillaries. This is bordered by an orderly transitional type of epithelium.

bordered by orderly rows of epithelial cells in palisade arrangement (fig. 3). In other areas the tumor has lost this regularity and the cells vary in size

FIG. NO. 4. Section of the tumor in which the epithelial cells show a distinct tendency to form alveolar or gland-like spaces. In such fields as this, there are numerous mitoses, but there is no evidence here of invasion or infiltration into the stroma.

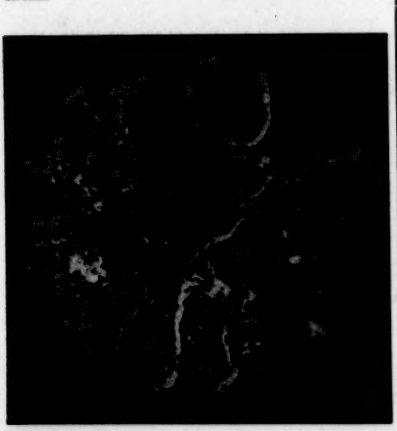

FIG. NO. 5. In this field the histological structure is more complicated. At the top is a nest of cells, orderly, and arranged in transitional formation as in Fig. 3. To the right and left, the cells show the same filamentous gland-like structures seen in Fig. 4. In the center there is a field of tumor cells, growing irregularly, showing little or no differentiation, and infiltrating the stroma and invading the lymphatics.

and shape; the nuclei are unusually large, mitoses both symmetrical and asymmetrical are common, and in spite of an existing papillary structure of the stroma the epithelial cells are growing in the most disorderly alveolar arrangement (fig. 4). There are spaces bordered by epithelial cells, filled with fluid containing very little protein. In some areas these cyst-like spaces contain mononuclear cells, single or in groups. Portions of the tumor which show this rapid growth, with marked anaplasia, show rather widespread areas of necrosis along the tips of the papillae. The stroma is composed of delicate, thin-walled vessels distended with blood. There are scattered foci of lymphocytes and mononuclear cells in the surrounding connective tissue stroma, and in some areas there is very widespread hyalin infiltration. The tumor in other areas is growing in still another manner, where expanding sheets of cells invade the stroma of the tumor and often arrange themselves to suggest pearl formation (fig. 5). These cells are quite large, vary in size and shape, have large nuclei rich in chromatin and frequently show both mitotic and amitotic nuclear division.

There is a chronic non-specific inflammatory reaction along the entire surface of the dilated pelvis with partial destruction of the epithelium and scattered calcareous incrustations. At many points the infection has spread up and into the tubules producing quite extensive reactions within and about the tubules leading to destruction of the tubules, and abscess formation. Although the inflammation may be traced histologically to the periphery of the kidney, there is no evidence that the infection in the surrounding fatty tissue has followed a direct extension from that of the kidney. The inflammatory reaction about the kidney with destruction of wide areas of tissue, thrombosis of both arteries and veins, hemorrhage, and necrosis, together with the formation of tracts of granulation tissue, was apparently the result of an infection spreading from the damaged and distended wall of the pelvis. There are still large areas of the kidney showing, with the exception of a slight swelling and granular degeneration of the cytoplasm of the epithelial cells of the convoluted tubules and little coagulated protein in their distended lumina, no pathological changes.

COMMENT

In a recent paper by MacKenzie and Ratner, the symptomatology of tumors of the renal pelvis is thoroughly discussed. Hematuria, pain, swelling in the region of the kidney, and the passage of tissue, comprise the more important urinary signs and symptoms. In the case herewith reported, there was at first only hematuria and pain in the left kidney region. No mass was palpable until obstruction of the ureter occurred. Pyelographic studies offered our most accurate aid to diagnosis. It was the filling defect shown in the pyelogram which enabled us to make a diagnosis of renal tumor, probably tumor of the kidney pelvis. The presence of blood clot in the kidney pelvis must always be considered as a possible cause of a filling defect of the type seen in this plate and in no way can a filling defect from the blood clot be definitely excluded in making a diagnosis. It is well recognized that the usual procedure in cases of papillary tumors of the renal pelvis is ureterectomy in addition to nephrectomy because of the frequency of secondary implantation along the urinary tract. In the case here reported, where the patient was definitely a poor operative risk, removal of the ureter was not considered justifiable. This case is of surgical interest in the following

respects: (1.) When the patient was first seen the tumor was not palpable, and a diagnosis of tumor was made chiefly on the filling defect shown in the pyelogram. (2.) A ball-valve occlusion of the ureteropelvic junction caused by the tumor led to a rather rapid development of an infected hydronephrosis and extensive pyelonephritis which demanded immediate nephrostomy. (3.) The dense inflammatory tissue surrounding the kidney and its pelvis made satisfactory palpation impossible and the kidney was removed on pyelographic evidence of tumor.

From the standpoint of pathological histology this tumor is of unusual interest because it shows that within such a nodule both benign and malignant areas may be found, and, secondly, that by metaplasia several histologically different types of epithelial tumor may develop, namely, regular transitional palisading, disorderly alveolar formation, and diffuse medullary areas, showing a tendency to pearl formation with invasion of the stroma.

SUMMARY

A case of papillary carcinoma of the kidney pelvis is reported with both clinical and pathological studies. The intermittent signs and symptoms, probably best explained by the action of a ball-valve occlusion of the ureter at the uretero-pelvic junction by this pedunculated papillary tumor and the unusual and rather complex histological structure of the tumor are of unusual interest.

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UTERUS DIDELPHYS

With Report of a Case

BY RICHARD H. SWEET, M.D.†

A. E. ROCKEY writing in 1916 published an article on double uterus and vagina in which he remarks, although stating as his personal opinion that this condition is more common than ordinarily supposed, that the literature until that date contained very little reference to it. According to him the first sixty volumes of the *Annals of Surgery* do not mention it. Parvin writing in Ashurst's System of Surgery thirty years previously treated of it and gives eight quotations from the literature. Kelly in his Operative Gynecology, published in 1898, gives an illustration of a case. In spite of this a survey of the literature reveals the fact that there had been considerable interest in the subject before, and indeed as early as 1705 Littre in the Histoire de l'Académie Royale des Sciences describes a case of true uterus didelphys in a female infant. The scientific merit of this description makes it of sufficient interest and value to quote it here. Littre says in part*, "M. Littre, en dissequant une petite fille morte à l'age de deux mois, trouva qu'elle avait le vagin partagé par une espèce de cloison en deux cavités égales, l'une à droite l'autre à gauche, de manière, cependant, que la cloison était entière et ne formait deux cavités absolument separées que depuis le milieu du vagin jusqu'à la matrice. Chacune de ces deux cavités aboutissait à une matrice particulière qui avait son orifice, son cou, son fond, le tout parfaitement séparé de la matrice voisine, mais parfaitement semblable en figure en consistance, en dimensions. Les deux matrices, depuis le cou jusqu'à une certaine profondeur, n'étaient que comme une seule partagé en deux par un cloison, mais leurs fonds étaient entièrement distincts et détachés l'un de l'autre. Chaque matrice n'avait qu'un ovaire et qu'une trompe, qu'un ligament rond et qu'un ligament large."

At the end of this careful and concise description of the condition observed in the case, one finds this interesting philosophical speculation illustrating the state of knowledge of that day:

> "Si tous les animaux ont étés immediatement formés par la main du Souverain Ouvrier, on ne peut guere s'empêcher de croire que ceux d'une meme éspèce ont été formes entierment semblables et que les configurations ou dispositions extraordinaires de parties viennent de quelques accidents fortuits du developpements des oeufs et les monstres du mélange de plusieurs oeufs. Mais comment cette matrice double ã-t-elle pu être l'effet d'un accident fortuit du developpement? Il est difficile de l'imaginer. Ces accidents peuvent détruire, déplacer, alterer quelques parties, mais pas en produire de nouvelles. Serait ce que deux oeufs femelles se seraient attaches ensemble et que toutes parties de l'un auraient peris excepté la matrice, qui, par consequent se serait

*Editor's Note: According to the manuscript.

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trouvee double sur le foetus resultant de ce mélange? Cette supposition parait un peu forcée, et peut-être, cependant n'y a-t-il rien de plus recevable."

In the literature since Rockey's publication, one finds increasingly frequent case reports of great interest and several articles dealing with the points of major importance from the standpoint of embryology, symptomology and especially the influence of the condition on parturition. Notable among these are those of Dubreuil-Chambardel, P. Findley, F. C. Newton, and N. F. Miller.

Many varieties of uterine anomalies resulting from partial or incomplete fusion of the Muellerian ducts are known. The embryological origin of all of these is now well understood and has been too often described in the literature to require any re-statement here. There is, however, still some confusion as to the terminology used to designate the various forms of bipartition of the uterus. The term *uterus didelphys* is used to designate the condition where there is complete failure of fusion of the Muellerian ducts resulting in total duplication of the genital tract with two uterine fundi, two cervices and two vaginae. By certain writers the term is applied only to the very rare cases which include in addition a bipartite vulva. But ordinary usage does not necessarily make this limitation. Lesser degrees of duplexity are to be referred to as uterus bicornis with or without a partial vaginal septum and with a single or double cervix; or in cases where fusion has fallen just short of normal, as uterus septus with or without vagina septa. Obviously all of these several degrees of partial or complete biparity of the genital tract are characterized by the presence of no more than two ovaries and two fallopian tubes. On the other hand, it is not uncommon, as in the case here reported, to find atrophic or absent tubes or ovaries on one or both sides in these patients.

A fact of interest which is brought out by a review of the cases reported in the literature is that uterus didelphys, as well as the lesser degrees of congenital biparity of the genital tract, is frequently associated with other curious congenital anomalies of development. In the author's case there was an abnormality of the anus and an incomplete rotation and fixation of the colon. Absence of one kidney, usually the right, has been reported several times. Patent urachus, double anus, the occurrence of a cloaca, and duplication of the bladder have all been reported.

Much has been written on the complications of this condition, especially the dangers which result from pregnancy and parturition. But a thoughtful observer must admit that dangerous complications are less common than ordinarily supposed and that many cases have been discovered by mere chance examination. Indeed many cases are on record where the presence of a didelphic uterus has not been detected until after one or two children have been born. The clinical importance of this condition, however, concerns chiefly the obstetricians. Abortion is frequent, occurring according to Dunning in 23 per cent of the cases as quoted by Findley. Labor is often prolonged because of weak uterine musculature. Rupture of the uterus is fairly common for the same reason and postpartum hemorrhage is said to be slightly more common than in the normal uterus. Although spontaneous delivery is the rule, one of the major obstetrical difficulties which may arise in these patients is obstruction of labor resulting from incarceration of the non-gravid uterus in the pelvis. This occurs as a result of rotation of the non-gravid uterus into the hollow of the sacrum as the gravid uterus enlarges. Because of this condition, cesarian section has frequently been performed, but as Findley points out, this possibility should not lead one to advise radical measures such as hemi-hysterectomy or elective cesarian section without a test of labor because of the natural tendency of these patients to deliver spontaneously. The patient whose case is reported here was delivered of four normal children without difficulty or prolongation of labor. Torsion of a pregnant horn has been reported by H. W. Horn. Streptococcic infection as a result of puerperal sepsis of the non-gravid uterus has been reported. For an excellent review of the obstetrical aspects of this condition the paper of Findley presenting the results from a study of 135 cases of uterus didelphys should be consulted.

Complications and associated lesions which occur as a result of uterus didelphys in the non-pregnant state, have been frequently described. Hematocolpos on one side is fairly common; hematometra also occurs as well as hematosalpinx, all the result of stenosis and occlusion of one genital tract at various levels. This condition has been treated by unilateral salpingectomy or hysterectomy. A case of gangrene of one uterus as a result of torsion has been reported. Fibroids may occur and carcinoma has been noted, but there is no evidence to show that these lesions are any more frequent in uterus didelphys than in a normal single organ.

Coitus in this condition is usually confined to one vagina and as a result of this, the other vagina remains small and often virginal. There are cases on record, however, of impregnation in both sides, either alternately as in the case reported by Davies and Cellan-Jones, or concurrently as reported by Garcia. One must, therefore, recognize the theoretical possibility in these patients of a true superfoetation and cases of multiple pregnancy of this sort have been reported. Patients with uterus didelphys

are said to be unusually fertile, although as noted above, there is a compensatory high rate of spontaneous abortions. One should note with regard to function that coition, pregnancy, and parturition commonly proceed perfectly normally and without ill effects in these patients. To recognize this fact is to save many of these women from unnecessary and ill-advised operative interference. To recommend surgical measures in these women with no other justification than the mere presence of this congenital anomaly cannot be justified on the basis of experience and to operate excepting for known pathological conditions is no more reasonable for them than it would be for a normal person.

CASE REPORT: Date of first visit November 4, 1931.

S. L. G., aged 29 years applied complaining of pain in the right lower quadrant of the abdomen.

PRESENT ILLNESS: Three months before entry she began to have attacks of pain in the right lower quadrant at first induced by lifting or bending over and followed by persistent dull aching pain in the same region in the intervals between attacks. Associated with this was the occasional occurrence of faintness and in addition a continual dragging pain in the pelvis similar to what she experiences during menstruation. She consulted her physician who referred her for diagnosis to a hospital clinic which started her on a round of visits to various hospitals each giving her somewhat different advice and contributing to her growing state of confusion and anxiety about her condition. The main tenor of opinion was to operate and remove part or all of her reproductive organs. During the week preceding entry she had been confined to bed with an ice-bag to her side which gave her some relief.

PAST HISTORY: The patient was born in New Hampshire and has always lived in New England. She has enjoyed excellent health until the past two years. There is a history of measles and pertussis and occasional tonsillitis while a small child. She was born with a single opening for her digestive and urogenital tracts. At the age of thirteen years she was admitted to a Boston Hospital where she was subjected to a series of three operations purporting to separate the digestive and urogenital tracts by constructing a new rectum and anus. Tonsillectomy was also performed while she was in the hospital. There is no history of injuries or of operations or hospital entries except as mentioned above.

The cardiorespiratory, gastro-intestinal, and neuromuscular systems have never been involved in any disease or been the cause of any symptoms.

Urinary History: There was enuresis until after the operation on the rectum and vagina, after which there was never any more urinary difficulty.

Catamenia: Her periods began at the age of thirteen years, regular every twenty-eight days, lasting four days, requiring four napkins a day. The last two periods have been prolonged slightly and more profuse, as a result, she thinks, of an examination under ether performed at another hospital. No metrorrhagia. She has always had some dysmenorrhea which consists of a sharp pain in the side of the abdomen with dragging pain in the back and lower pelvis.

MARITAL HISTORY: The patient has been married thirteen years. Husband is alive and well. There are four children alive and well. Two miscarriages; one, the first pregnancy at seven months following a fall; the other which was the third pregnancy, at four months after being thrown down a flight of stairs. She was ill for several weeks after each miscarriage. All four children were full term, breech deliveries, the last one a difficult one requiring high forceps.

PHYSICAL EXAMINATION: The patient was in a state of great anxiety about her health. The general examination revealed nothing remarkable except carious teeth. There was tenderness in the right lower quadrant of the abdomen but no rigidity. Examination of the vulva revealed a double vaginal orifice, each side leading into a separate vagina, both vaginae lying side by side in a transverse plane. At the apex of each vagina was a perfectly formed cervix, the left much smaller than the right and nulliparous in appearance. The right cervix was moderately lacerated transversely as a result of pregnancy. Each uterus could be felt bimanually. That on the right was about the size of a normal fundus and in addition there was apparently a smaller very tender elongated mass attached to it. The left uterus felt small and was not tender. The anus was very close to the double introitus and the sphincter was apparently incomplete anteriorly, this portion being replaced by a thin band of tissue at what is normally the fourchette. This condition represents the end result of previous operations to form a separate anal opening.

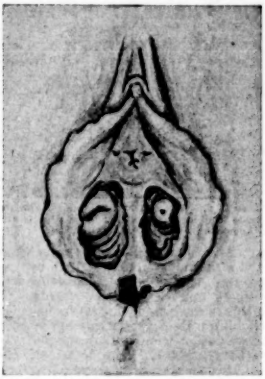

FIGURE NO. 1. Appearance of the vulva showing double introitus, vaginal septum and separate cervices.

DIAGNOSIS: A preoperative diagnosis of uterus didelphys with right chronic salpingitis was made.

OPERATION: November 19, 1931.

On examination under ether anesthesia it could not be determined whether the mass on the right side was only the uterus or whether it was partly tumor. A laparotomy was therefore done through a right paramedian incision. The gallbladder was explored and found to be negative. The colon was abnormally rotated, the cecum pointed upwards and the appendix extended up toward the liver. From this point the transverse colon extended obliquely downward toward the left side of the pelvis where there was a very redundant sigmoid which ended in the rectum, which seemed to disappear between the

two uteri. It was noted that the colon had a very long transverse mesocolon so that the colon hung down much longer than the great omentum which was not adherent to the transverse colon as it is normally, but hung from the stomach as a separate sheet. The uterus on the right was about normal size for a woman who has had children. It had one tube extending from the cornu. The ovary on this side was normal. The uterus on the left side lay down along the side of the pelvis and was considerably smaller than that on the right. It was about the size of that of a girl of 16. It had a rudimentary tube which was adherent to the posterior and lateral surfaces of the pelvis and which faded out in the peritoneum. The only sign of an ovary on this side was a small nodular mass about 1 cm. in diameter. It is questionable whether this rudimentary ovary is functional. The right tube was removed. The left tube was divided and tied on either side. The right uterus which had a marked tendency to prolapse into the pelvis was suspended by a modified

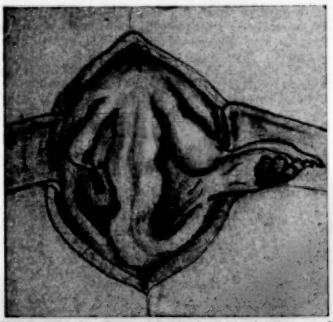

FIGURE NO. 2. Intra-abdominal view showing condition of uteri, tubes and ovaries. Right tube and ovary shown as withdrawn from the abdomen for inspection.

unilateral Olshausen suspension, suturing the round ligament up to the abdominal wall by means of a suture passing through the rectus sheath and the rectus muscle and tied on the inside. The space between this suture and the lateral peritoneal surface was obliterated by means of a stitch. The appendix was then removed by means of a cautery and the stump inverted. The colon was then placed down into the pelvis so as to avoid contact of the small intestines with any raw surfaces. (Fig. 2.)

POSTOPERATIVE DIAGNOSIS: Uterus didelphys, atresia and hypoplasia of the left tube, hypoplasia of the left ovary, congenital abnormal rotation of the colon, congenital anomaly of the great omentum.

Convalescence was uneventful and healing was complete; the patient was discharged at the end of two weeks.

Follow-Up Notes:

January 20, 1932. Feels much improved; better than for many months. Complete relief from pain.

August 23, 1932. Very well; no pain or discomfort of any sort. Menses normal and relatively free from pain.

February 3, 1933. Still very well. No symptoms.

COMMENT

There are certain aspects of this case which are of interest. As is so often true, there were other congenital anomalies, in this case of the gastro-intestinal tract. Incomplete rotation of the colon appears to be one of the most common associated conditions. I have operated on another patient with this type of colon who also had a uterus bicornis. The lack of attachment of the great omentum to the colon is of passing interest. The anomaly of the anus which was present in the case presented is less common. Details about this condition during childhood when she had some sort of a plastic operation are lacking, but there was apparently a cloaca-like arrangement similar to what has been reported by other observers.

Coitus in this patient was always effected through the right vagina and all six pregnancies were in the right uterus. In view of the findings at operation, an element of interest arises from the concept that this woman is provided by nature with a device for the prevention of conception. The hypoplastic imperforate tube on the left would make the effective entrance of spermatozoa impossible after coitus through the left vagina. On the other hand, as she has demonstrated, coitus through the right vagina resulted readily in impregnation.

Significant in this case is the fact that all of the surgeons whom she consulted had unnecessarily radical ideas regarding treatment. Duplexity of genital organs is normal to this individual and not in itself a cause for symptoms and much less a reason for radical surgery such as extirpation of one side with division of the vaginal septum, as advised by the majority. The truth of this statement is borne out by the fact that gestation and parturition, save for two miscarriages each following a fall, were uneventful and also by the return to a perfect state of health following the exercise of conservative surgical procedures. To this woman the assurance that it was unnecessary to do a partial or complete hysterectomy restored her emotional balance, and it is reasonably certain that, had she been subjected to this procedure, she would have become a nervous invalid.

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RECURRENT TRICHOBEZOAR*

Report of a Case

BY DARWIN E. BENNETT, M.D.†

CASES of "hairball" or trichobezoar are unusual and warrant reporting. This case presents several interesting features. In the first place, it is an instance of recurrent trichobezoar, the first hairball having been removed twenty years previously. The only other case appearing in the literature in which there was recurrence was reported by Harris[1] in 1925. In this instance the bezoar reformed and was removed six times in thirteen years. The one recorded here was not recognized until twenty years following the removal of the first hairball.

In the second place, this patient is fifty years old, the oldest patient reported to date. The next oldest was Gemmel's[2] case, an insane man of forty-three. The great majority of cases reported are girls and young women.

The third interesting feature is the chronicity. Although the patient had undoubtedly carried the tumor for many years, careful questioning failed to elicit any history of gastro-intestinal symptoms except marked constipation. The tumor was discovered only when she came to the hospital because of another complaint.

Case Report

A fifty-year-old, native-born, white woman entered the Peter Bent Brigham Hospital on February 13, 1933, because the daily trips to the Outdoor Department for the dressing of a large gluteal abscess were too great a drain on her generally debilitated condition. She is the mother of three children, two of whom died in infancy, and she had four miscarriages between pregnancies. She has worked hard all her life at tasks involving a great deal of drudgery. Twenty-five years ago she worked for several months in a shop where she made wigs and

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"switches". Twenty-one years ago she entered the Brockton Hospital, Brockton, Massachusetts, because of an attack of nausea and vomiting. At an exploratory operation a hairball which weighed two pounds was removed from her stomach. At that time she gave a history of having lost her hair six years before. (Most of the past history was obtained from the records of the Brockton Hospital after the patient had reluctantly admitted having been treated there.)

For six years prior to her recent hospital admission, she has been troubled with rheumatism, falling of the womb, and intense itching. About one year ago she experienced a low-grade chronic pain in the region of her left hip and eight months ago a lump appeared in the left gluteal fold. It was

FIG. 1. Roentgenogram. The appearance of the "hairball" when outlined by the opaque meal. Note the projection into the antrum of the stomach and the absence of an air bubble in the fundus.

somewhat tender and bothered her in getting up and down. Gradually it increased in size and on the day of entry to the dispensary it broke spontaneously, discharging about a quart of pus. The abscess was debrided under gas-oxygen anesthesia. After two visits for dressings of the indolent infection, the patient was unable to make the daily trip and was sent into the hospital for observation.

Physical examination: The patient was an elderly, emaciated, white woman with thin brown hair turning grey. Her facies was pale, haggard, and apprehensive. Her psyche was apparently normal, though her intelligence quotient was low. The skin was blotchy with many small, brown pigmented areas and acne-like scars, more numerous over the back. The hair was pediculous and there were lesions typical of scabies on the hands and thighs. The mucous membranes were pale and atrophic. Examination of the chest showed the diaphragm high and relatively fixed on each side. A hemic murmur was heard all over the precordium. Her blood pressure was 110 systolic over 75 diastolic. The abdomen was asymmetrically rounded and slightly distended by a large, hard, movable, and non-tender mass which filled the entire left upper quadrant and presented a definite notch along the right border just above the umbilicus. The mass was interpreted as an enlarged spleen. The region of the left great trochanter was quite markedly tender to palpation and there was limitation of abduction of the left hip. The fingers showed the lesions of atrophic arthritis. There was a 7 x 5 x 5 cm. defect lined by granulation tissue in the left gluteal fold. She weighed 100 pounds and her temperature was 100.2° (F).

Laboratory data: White blood count was 13,500; hemoglobin was 40% (T); red blood count was 3,800,000. A differential count (Wright's stain) showed 80% polymorphonuclears, 19% lymphocytes,

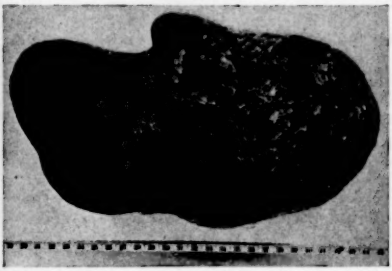

FIG. 2. Hairball. Weight, 1720 Gm. Measurements, 25x14x8 cm. Note the projection on the left border which filled the antrum. Above the projection is the "notch" palpated on physical examination.

and 1% basophils. The blood-smear showed marked achromia and slight variation in size and shape of the red cells. The platelets were normal. A gastric analysis both before and after operation showed no free hydrochloric acid. No foreign material was noted in the specimens. Urine, stool, and serology negative. A culture from the abscess cavity grew Staphylococcus aureus. A guinea pig inoculated with curettings from the abscess cavity was negative for tuberculosis.

Roentgenograms of the left hip showed calcification in the soft tissues but no bone involvement. A barium enema showed no downward or right displacement of the splenic flexure, as would have been present with a spleen as large as the mass palpated. When the patient drank barium it was seen to hesitate at the cardia and then trickle slowly down, over, and around a large non-opaque foreign body which completely filled the stomach and extended into the antrum but not through the pylorus. This was interpreted by Dr. M. C. Sosman as a trichobezoar. (Fig. 1.)

Hospital course: On February 24, 1933, Dr. E. C. Cutler removed the bezoar through a left transverse abdominal incision and a 20 cm. incision in the fundal region of the stomach. The specimen measured 25 x 14 x 8 cm. and weighed 1,720 Gm. (3.78 lbs.). It consisted of fine-textured, dark brown hair firmly matted together. (Fig. 2.) The bezoar is now in the Warren Museum at the Harvard Medical School and after three months' desiccation weighs 550 Gm. (1.2 lbs.).

The patient made an uncomplicated recovery from the operation but in the three months since the removal of the bezoar the infection in the left gluteal region has not cleared up and the anemia has improved only slightly. Repeated attempts were made to elicit from this patient a confession of swallowing her hair, but we were unsuccessful. The

patient was unquestionably ashamed of the habit and though she denied the direct accusations, she suggested that she might have eaten her hair in her sleep. Following the operation she was seen on several occasions pulling out her hair, but her "shyness" made it impossible to catch her actually putting hair in her mouth.

SUMMARY

This instance of trichobezoar is noteworthy because it represents a recurrence, because of the patient's age (fifty years), and because of the probable absence of any gastro-intestinal symptoms over a very long period.

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THE SYMPTOMS OF HIDDEN OCULAR MUSCLE IMBALANCE

BY F. W. MARLOW, M.D.*

LACK of appreciation of the symptoms which may be due to muscle imbalance and particularly to hidden muscle imbalance (latent heterophoria), accounts for many of the numerous patients who suffer year in and year out from headache, gastric disturbance, neurasthenic and psychasthenic symptoms in spite of the fact that they have consulted both general and special physicians of high rank. This lack of appreciation on the part of the profession is partly due to the fact that textbooks, both of general medicine and ophthalmology, fail to give the attention to the subject or to lay the stress upon it which its importance justifies. In fact neither in the general nor special literature does the subject receive the consideration due to it. But it is hard to credit the statement of a recent writer that, "a routine muscle examination is seldom made even in private practice." Just how true this is the present writer is in doubt, but it is certainly true that if the motor apparatus of the eyes were accorded the same detailed attention which the refraction commonly receives, the failures to obtain relief would be fewer. Just as it is essential to measure not only the manifest but also the latent refractive error, so also is it essential to measure not only the manifest but also the latent muscle imbalance.

No doubt Bielschowsky is correct in stating that headache and other asthenopic symptoms cannot be explained by anomalies of refraction and motility alone. Predisposition of some kind, sometimes acquired, more commonly inherited, is present in determining whether the individual can carry the load and show no symptoms, or by lowered resistance will break down under it, and consequently come under observation for any of a group of symptoms of the neurasthenic type. In many of these cases improvement in general health, by whatever means, may eliminate the symptoms, at any rate for the time being, but in many others all treatment fails until both accommodative and particularly any muscle imbalance strain is removed.

There are, therefore, three factors requiring consideration, namely, the refraction, the muscle balance, and the general health.

The influence of refractive error can be eliminated with a high degree of certainty because with the aid of cycloplegia an accurate measurement of it can be made. When competent medical advice has also failed to effect relief by improving the general health, it may be assumed that further efforts in that direction are futile. The only factor remaining for investigation is the muscle balance.

The 100 consecutive cases upon which the present paper is based are those left over, so to say, after repeated attempts at relief by correction of refractive errors and manifest muscle imbalance, and by treatment of the general health by physicians have failed to relieve or have aggravated the symptoms.

In other words these are cases in which the influence of all refractive error and manifest imbalance has been eliminated, the same being true of the influence of the general health, yet the symptoms, apparently those of eye-strain, persist. This clears the field for a more intensive investigation of the latent phorias and their bearing on the symptoms complained of. It may be proper to state here precisely what is meant by a latent phoria.

The common tests for muscle imbalance, or heterophoria, depend on the temporary annulment of binocular vision by covering one eye with a card, the other eye fixing a light at 6M distance, shifting the card to the other eye and noting whether the eye remains stationary and whether the patient sees any apparent movement of the light. If the eye and the light remain stationary then no imbalance is demonstrated or vice versa. This and similar tests based on the same principle are of short dura-

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tion. It has been found by experience that they are not long enough to permit muscle relaxation to take place and therefore that they often do not reveal the whole error or even the kind of error. A prolongation of the cover test for a week at least is necessary to effect a relaxation of the muscles and to show the kind and degree of imbalance. In other words the cover test, which is generally recognized as the most satisfactory, is simply extended in point of time.

The writer has gone over his records of these 100 consecutive cases in which repeated correction of the refraction and attention to general health have failed to relieve the symptoms or have aggravated them, and noted the symptoms recorded in each case. In these cases a prolonged occlusion test finally revealed important errors of muscle balance, the correction of which afforded the desired relief in a majority of them. It is therefore fair to assume that the symptoms were due to latent deviation undemonstrable by the ordinary short tests. The frequency of the more important symptoms is approximately shown by the figures accompanying them. The other symptoms noted occur with far less frequency but nevertheless are worthy of consideration, and their persistence in spite of previous treatment may constitute a presumption of an undemonstrated strain.

These symptoms may be divided into three main groups, (1) local or ocular (2) disturbances of digestion and assimilation and (3) functional disturbances of the nervous system. It is quite common to find that many patients present symptoms not of one group only, but often of all. Thus one of the commonest groupings is that of headache, photophobia and indigestion. Photophobia indeed is so commonly present, in association with other symptoms, that its absence lessens in one's mind the expectation of finding a latent phoria; but it may be absent. It is recorded as having been present in 61 of the 100 cases, but this probably understates its frequency. The same is true of the headache and asthenopia, respectively noted in 64 and 68 cases. Gastric disturbances of one kind or another occur in 69 cases.

The following list shows the great variety of symptoms which may be caused by hidden muscle imbalance.

1. Local or Ocular	Styes	
	Chalazion	
	Chronic conjunctival congestion	
	Blepharo-spasm	
	Twitching	
	Blinking	
	Photophobia	61 cases
	Asthenopia	68 cases
	Aching, etc. / Smarting, burning	27 cases
2. Stomach	Indigestion	24 cases
	Nausea and Vomiting (with or without headache)	44 cases
	Anorexia	
	Faulty Assimilation	
	"Bilious" Attacks	
	Cyclical Vomiting.	
3. Nervous System	Headache	64 cases including neck and back in 18 cases
	Head not clear	
	Confusion in head	
	Irritability	
	Nervousness	
	General Fatigue	
	Vertigo	8 cases
	Mental Inefficiency	
	Psychoneurosis.	

Group 1

The two most outstanding symptoms in this group are asthenopia and photophobia. For the asthenopic symptoms, repeated examination and refractive correction have been made without success. Many such patients state that they have to remove the glasses on account of the increased strain caused by them and after a while have to replace them because they cannot see without them or have some other type of discomfort. They are not comfortable either with or without the glasses. This may be interpreted as meaning that without glasses there is accommodative strain, and that with the glasses, which correct the refraction, the muscle strain is aggravated, presumably because the better defined retinal images require more exact superposition and consequently the demands on the extrinsic muscles are greater. As previously stated photophobia is present in a large percentage of the cases, is commonly of lifelong duration and may vary in degree from slight discomfort to a degree of intensity necessitating the constant wearing of smoked glasses. The most severe and troublesome cases of photophobia seen by the writer have been due to this cause. These patients often come wearing smoked or tinted glasses, and if they do not actually scowl present a characteristic mid-frontal vertical furrow just above the root of the nose, in itself alone very suggestive of muscle imbalance. The other manifestations named are of less frequent, but still somewhat common, occurrence.

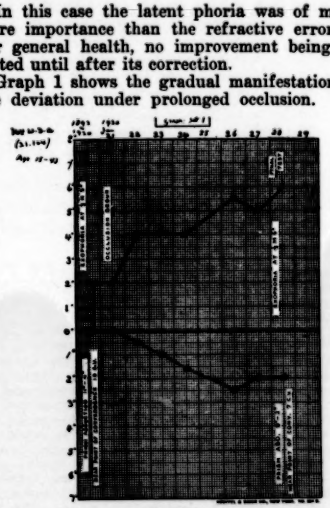
The two following cases belong to this group.

Case 1. Mrs. W. D. A., wife of physician. (21-204.) With *styes, headache* and *asthenopia*. Under care for asthenopic symptoms since the age of 15. (1892.) Then the right eye was emmetropic and the left showed hypermetropic astigmatism 0.25D (under cycloplegia). Orthophoria. Slight changes were made in her correction from time to time until 1919. From April of that year until January, 1920 she suffered from a succession of styes which recurred in spite of the use of yellow oxide of mercury ointment and other treatment. Headache and insomnia were also present. Her refraction on Jan. 21, 1920, was

R +0.50s +0.25c 80°, L+0.50s. Tests showed exophoria 2°. An occlusion test begun on that day and continued for seven days brought out left hyperphoria 2° and exophoria 6°. She was given right eye 1½° prism base in, L 2° base in down 30°, in addition to the refractive correction, and a note in January, 1923 stated that there had been no recurrence of styes.

In this case the latent phoria was of much more importance than the refractive error or her general health, no improvement being effected until after its correction.

Graph 1 shows the gradual manifestation of the deviation under prolonged occlusion.



GRAPH 1.

In this graph and the following, "X" when placed above the zero 0° line indicates divergence (exophoria), when below it convergence (esophoria), when on the 0° line no lateral deviation.

"O" when above the zero line indicates a deviation of the *right* eye upward (R hyperphoria), when below it a deviation of the *left* eye upward (L hyperphoria). When on the zero line that the eyes are on the same level.

CASE 2. Mrs. F. O. (59:129.) Severe *photophobia* and *asthenopia*. Under observation since age of 33 on account of headache and other asthenopic symptoms and photophobia. Examined and prescribed for from time to time until the age of 57, at which time the photophobia became extremely distressing and was unrelieved by refractive correction (low hyperopic astigmatism) or other treatment. At every examination up to this time she had shown a perfect muscle balance (orthophoria). At the end of a 16 day occlusion test she showed R Hyperphoria 3° and exophoria 1°-3°, and a 2° prism base down for her right eye added to her refractive correction gave prompt and complete relief.

In this case nothing was accomplished until the hidden muscle imbalance was demonstrated and allowed for in the glasses.

GROUP 2

Stomach symptoms vary from chronic so-called nervous indigestion to severe gastric upsets either in association with headache or alone.

Nausea may be directly associated with use of the eyes.

Cyclical vomiting in children or young adults which may be regarded as a variant of sick headache.

That assimilation is interfered with is mainly shown by the increase in weight which often follows the correction of a phoria.

CASE 3. Mrs. L. P. M., aged 33. (641.) *Indigestion, headache, photophobia—impaired assimilation.* Seen March, 1911, the subject of headache since childhood, constant for the past five years, in temples occiput and vertex, not accompanied by nausea. For the same period she had been the subject of intractable indigestion which has resisted all effort at treatment. She was running down in general health. A sister had died following a similar group of symptoms and "malnutrition," and her friends thought she was going the same way. Glasses had been prescribed which were an approximately accurate correction of her refraction (hypermetropic astigmatism) which aggravated her headaches though she could not go without them. Tests for muscle balance showed exophoria 6° and *right* hyperphoria less than ½°. A 2° prism base in was added to each lens and gave some temporary relief. At a later date an occlusion test lasting eight days brought the exophoria up to 14° and revealed *left* hyperphoria 2¾°. Glasses correcting 7° of exophoria and 2° of hyperphoria gave complete relief from the symptoms for a time. A little later partial tenotomies of the left superior rectus and right externus were done. During the next six months she increased 15 pounds in weight and although she has had some occasional headache and indigestion she has called herself well ever since. Twenty-one years have elapsed since the operations.

This case emphasizes very forcibly the futility of general medication and refractive correction in the presence of a latent and uncorrected phoria. It shows also the influence of the latter on assimilation.

CASE 4. Miss H. V., aged 20. (A9240.) *Cyclical vomiting.* Seen December 12, 1932. A history of vomiting spells in childhood which ceased in high school, and did not recur until the spring of 1932 when she had what she called a bilious attack. She was wearing at that time a 1½° prism base in over each eye. Since the omission of these prisms two months ago she has had attacks of uncontrollable vomiting preceded by pain in the right eye. The symptoms did not yield to general treatment, which included an appendectomy. Vomiting recurred a week after operation and her physician then advised examination by an ophthalmologist, the previous glasses having been prescribed and furnished by an optometrist. These lenses were +0.25s+0.25c 180° each eye. Examination under cycloplegia showed in each eye emmetropia, or very low hyperopic astigmatism, low esophoria, no hyperphoria. In other words the result of the examination was practically negative. An occlusion test was then done and showed that she had a deviation of the left eye upward (left hyperphoria) varying from 1½°-3½°, and no lateral deviation. A 1½° prism base down before the left eye was prescribed. Seven weeks later she writes, "I am feeling just fine," "no headache" and "no trouble with my stomach."

In this case also the influence of general medical and surgical treatment, and refractive error correction had been eliminated, leaving muscle imbalance as the most probable source of the trouble. This was confirmed by the prolonged

cover test and the correction of the error so found.

CASE 5. Mrs. L. C. W. (A8377.) Aged 25. *"Bilious" attacks, photophobia* and *asthenopia.* History of injury to head from fall from bicycle at the age of 10. When first seen in June, 1931 she complained of "bilious" attacks followed by headache for the past five or six years, recently occurring every week, for which she had received no benefit from medical treatment. She was wearing glasses which were an approximate correction for her refraction. Under cycloplegia she accepted R+0.50s, L+0.25c axis 85°. Muscle tests showed a normal balance. As such errors as she showed were quite inadequate to explain her symptoms, and as the latter were very suggestive of eyestrain, she was put through a prolonged occlusion test lasting eight days. At the conclusion of the period there was a divergence of 6½° and a deviation of the left eye upwards of 3°. Prisms, correcting 2° of the hyperphoria and 3½° of exophoria, combined with her refractive error gave prompf relief, which is maintained after 18 months. She has had no attack since.

Here again attention limited to general health and refraction completely failed to accomplish anything, and the sequel definitely proved that the latent phoria was the cause of the trouble.

GROUP 3

Disturbances of the nervous system may be grouped under the general term of neurasthenia or nerve exhaustion and consequently may present symptoms which vary with the individuality and temperament and occupation of the patient.

They include quite commonly a tendency to rapid fatigue, not only ocular but general physical and mental, nervous tension, irritability, making the patient "difficult to live with," vertigo, etc.

But the symptom for which advice is most commonly sought is headache, which is recorded as present in 64 cases.

The main characteristics of the headache are as follows:

1. It dates usually from an early period in life, sometimes being represented at the earliest period by cyclical vomiting. This excludes later more acute conditions such as sinus trouble, as possible causes. The cause must precede the effect.
2. Its location is commonly the frontal and occipital regions, sometimes the occipital region alone or in association with neck symptoms, rarely the frontal region alone and still more rarely the vertex. Pain, stiffness or discomfort of some kind in the back of the neck is recorded in 18 cases, but that certainly understates its frequency. It is one of the most constant and characteristic symptoms in these cases and is about as suggestive of muscle imbalance as photophobia is. It is certainly far more commonly due to this cause than to accommodative strain. It is often constant and it is not uncommon to see it cease promptly and completely on occlusion of one eye, even when it has been present for years or even from childhood.
3. Its frequency varies from daily or constant headache to periodical headache of migrainous type with varying intervals of freedom.
4. Its severity varies from moderate dull headache to pain of the severest type, putting the patient in bed for two or three days at a time, completely incapacitating him from work.
5. It may be accompanied by nausea and uncontrollable vomiting or be quite free from gastric symptoms. Sometimes the gastric symptoms are the most prominent, and the attacks may be called "bilious attacks."
6. The time of onset is commonly on waking but may be at any time.

The following cases briefly reported exemplify types of this group.

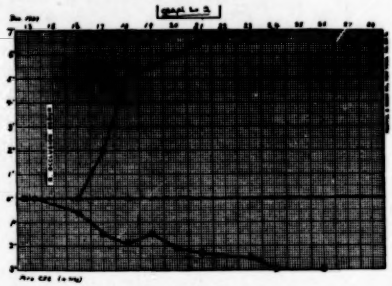
CASE 6. MRS. C. E. C. (A7196.) Aged 43. Wife of physician. Seen December 13, 1929. *Sick headache, photophobia* and *general fatigue.* Headache began in twentieth year, her first year of training as a nurse, and occurred at every menstrual period and sometimes in between, lasting from 5:00 P.M. or 6 P.M. until next A.M. after breakfast, accompanied by severe nausea and vomiting. Sometimes nausea occurred without headache. These headaches were unilateral, the pain being most intense in the eyes and back of the neck. Each headache was accompanied by a loss of six or seven pounds in weight. There were also spells of exhaustion without headache lasting three or four hours. She had worn glasses for 22 years without any influence on the symptoms, the last examination being one year ago, when no change was made. She was wearing R +0.75c 110°, L +75c 115°, approximately an accurate correction. Tests for muscle balance showed orthophoria. No hyperphoria could be detected in any part of the field.

A prolonged occlusion test (R eye occluded) lasting eleven days revealed a left hyperphoria of 3° and exophoria 7°. Prisms correcting 2° of hyperphoria and 4½° of exophoria were added to the refractive correction. In August, 1930 she reports herself as much better, less headache, slight nausea, much less exhaustion and has gained weight. On December 30 she reports no headache between Easter and October, some moderate ones lately and has gained 25 pounds. April 3, 1933, she reports total gain in weight 27 pounds and an occasional headache.

Graph 2 shows the gradual manifestation of the deviations.

CASE 7. T. W. D., aged 35 years. (7635.) University professor. *Asthenopia, neurasthenia, psychasthenia, photophobia, extreme nervous tension.* Seen in July, 1919, wearing R +0.75c 90° and L +0.62c 50°. Approximately accurate. Complaining that use of his eyes makes him so nervous all over that he "can't do a thing," that the last glasses strain his eyes and that he will have to give up his position, if he can't obtain relief. After cycloplegia R accepted +0.25 +0.87c 80°, and L +0.25s +0.75c 65°, and tests for muscle balance showed left hyperphoria 1° and esophoria 4°. A seven day occlusion test resulted in left hyperphoria 2½° and esophoria 16°. Partial tenotomies of both interni

and the right inferior rectus were done after a period of temporary relief by prisms. There was an immediate alleviation of the symptoms enabling him to continue his work, with gradual improvement.



GRAPH 2.

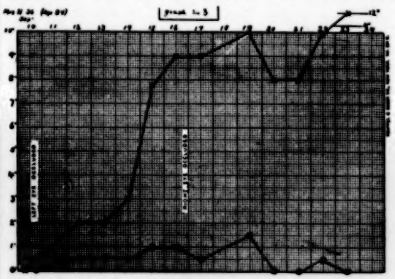
When seen last in October, 1932 he reported that he has been quite comfortable until within a few weeks. Examination showed that he required some modification of his refractive correction and more presbyopic correction.

CASE 8. G. M. (A4876.) Lecturer on psychology. Extreme *asthenopia, relative mental inefficiency.* This man had suffered all his life from asthenopia and photophobia with some intervals of comparative freedom from symptoms. The symptoms had been intensified during the seven years preceding his coming under observation, in the last two of which he was so incapacitated that he had to have his work read to him. During these seven years he had consulted nine oculists, and other specialists, all of national reputation, without relief. A seven day occlusion test brought out 6° of exophoria and 1¼° of left hyperphoria, the partial prismatic correction of which gave complete relief. This patient was kind enough to write out a full history of his case, which is published in his own words in the *Archives of Ophthalmology*, Vol. LVII, No. 4, 1928, and is referred to here because at a later date he published a book on the history of psychology and assured the writer that the part written after his correction was incomparably superior to the earlier portion, showing the influence of a latent phoria on mental efficiency.

CASE 9. MRS. E. F. H. (A9084.) Consulted me first on September 8, 1932, on account of seeing "specks" but gave a history of having been run down for one and one-half years and apparently having a nervous collapse in August, 1931.

She had seen various doctors during this time and in November, 1931 went to the Syracuse Psychopathic Hospital on account of extreme nervousness and various ideas. She was disturbed about the question of free will, had some thoughts of suicide, thought she had lost her soul and various other bad things about herself. She was very irritable and her neighbors annoyed her. For seven months she was unable to sleep even with sleeping powders. At the Psychopathic Hospital nothing definitely wrong was found although Dr. H. A. Steckel, the chief of staff, said that a tentative diagnosis of dementia precox was made. After leaving she became desperate, had no interest in anything, even in her son, and wanted to go back to the hospital. Later she went voluntarily to the Marcy State Hospital for three months, at the end of which time she realized that she was not insane and returned home.

Examination of her eyes showed a low degree of astigmatism in each eye, scarcely enough to be worth correcting or to be responsible for any nervous symptoms. It should be added that the symptoms pointing to eyestrain were intolerance of light, slight blurring in the distance and headache, which occurred daily, and occasional nausea. Her muscle balance was quite normal. No lateral or vertical deviation could be detected. Cycloplegia revealed no accommodative spasm. An occlusion test was begun on the same day the cycloplegic was used (September 10). There was a rapid development of divergence. The deviation went to 9° on the sixth day. Seven days later it amounted to 12°. During the period of the occlusion test she showed a varying amount of deviation of the right eye upward. (See Graph 3.) The final test, however, showed the



GRAPH 3.

eyes to be on the level. The exophoria was partly corrected with prisms, 2½° base in, each eye, the result being complete relief from all her symptoms.

She is well, is not nervous, does not worry about the question of free will or her soul and has put on flesh. Describes herself as being "perfectly fine".

This is only one instance of many in which a latent phoria proved to be an important factor in the production of mental disturbance, but it is the most extreme.

CONCLUSION

These cases and many similar ones in a series of about 2000 occlusion tests have forced the writer to the conclusion that eyestrain cannot be acquitted of responsibility for symptoms of the various kinds here described without a carefully carried out prolonged occlusion test. Although it is an annoying thing to do, patients of sufficient intelligence to understand the object of the test, when it is explained to them in a simple manner, accept it very readily, especially when they are the subjects of distressing symptoms which previous treatment has failed to relieve. As a matter of fact most patients become interested in the test as it proceeds and express willingness to carry it out as long as necessary.

MEDICAL PROGRESS

PROGRESS IN NEUROLOGY, 1932

BY ABRAHAM MYERSON, M.D.,* AND JULIUS LOMAN, M.D.*

EPILEPSY

In last year's "Progress in Neurology", we reviewed briefly Temple Fay's ideas on the production of epileptic convulsions, among which was the theory of deficient water absorption from the subarachnoid space. Stanley Cobb[1] takes issue with this theory. He does not believe that accumulation of fluid, sometimes found in epileptic brains, can occur from "supracortical increase in pressure" resulting in cortical atrophy. Cobb explains that since the flow of cerebrospinal fluid is from the ventricles to the subarachnoid space where it is absorbed, the pressure in the ventricles must be higher than in the subarachnoid space, otherwise there would be no flow. If now obstruction to absorption occurs (arachnoid villi and small venules and capillaries of the subarachnoid space), the flow of fluid will be slowed, but so long as any absorption occurs, the flow will continue and the ventricular pressure will continue to be higher than that in the subarachnoid space. If the obstruction were complete, the flow would cease and the pressure in the subarachnoid space would become equal to that in the ventricles. Cobb points out that there is no known mechanism by which the subarachnoid pressure could become greater than in the ventricles.

Cobb also doubts Fay's statement that water-drinking raises intracranial pressure in epileptics. The remarkable adaptability of the kidneys in maintaining the constancy of the composition and the volume of blood makes one doubt Fay's idea. This is borne out by Fremont-Smith's study of 15 epileptic patients. These were given 1200 cc. of water in three hours, the spinal fluid pressure being measured frequently. He found that in only three of the epileptics was there any delay in water excretion and in only one of these three did a convulsion occur.

Cobb believes that although restriction of water, which Fay advocates, may diminish the number of attacks, the direct experiments of Fremont-Smith and also those of Lennox show Fay's assumption as to the influence of water-drinking on the cerebrospinal fluid pressure to be erroneous. Cobb lists 56 clinical cases of fits and discusses their physiological mechanisms. At present, awaiting further research, Cobb reduces the numbers of mechanisms by saying that it seems probable that embryological defect and tissue destruction act by altering neural conduction. He points out the importance of cerebral anoxemia (this includes 36 of the 56 causes given). Hydration and dehydration probably act through chemical changes. Many drugs and chemicals cause convulsions by some unknown mechanism.

The Treatment of Epilepsy

Meggendorfer[2] used a combination of phenobarbital and belladonna in 37 cases of epilepsy and found a reduction of 33 per cent in the number of attacks as compared with periods in which only phenobarbital was used.

Hanns Manitz[3] treated 28 epileptic children with a combination of luminal and caffeine in a proportion of two to one and found that better results were obtained in over half the cases than by the use of luminal alone. He believes that the "hypnotic effect" of luminal is neutralized by caffeine and that the vasodilator action of the latter drug helps to counteract convulsions. (Our own clinical experience confirms this and we believe caffeine to be a valuable adjunct to sedative therapy.)

According to the experience of O. Beck[4], the ketogenic diet does not accomplish more than the other methods of treatment in epilepsy. Moreover, he states that the treatment is not free from harm, for he believes it is possible that a diet deficient in carbohydrates for long periods may result in acute loss of function of the insular apparatus of the pancreas. A contraindication, too, is that the diet is expensive and involves great difficulty, since it has to be given for long periods.

MIGRAINE

In a very long article, Henry Alsop Riley[5] discusses the various theories of migraine, including the reflex, "central", allergic, duodenal stasis, hypophyseal, toxic, endocrine, vasomotor or vegetative.

The reflex and "central" theories are disregarded as important. Although the allergic, duodenal stasis, and toxic theories may play a rôle in some cases, they leave unexplained the essential mechanism of migraine. The rôle of the glands of internal secretion in the production of migrainous attacks is indicated by the results of ovarian hormonal medication and by other clinical data. There is some evidence that some patients present changes in hormonal balance.

It is the failure of the vasomotor apparatus,

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however, which may be most plausibly credited with the production of migraine. Although the evidence is scanty, the surgical results are uniform in the relief of the headache by interruption of the afferent or efferent flow of impulses. How the perverted activity of the vegetative nervous system is brought about, resulting in vasomotor unbalance, is unknown. The hypothesis that a perverted hormonal balance is the inciting agent and causes a disordered vegetative nervous system seems to be the most tenable theory at present.

The surgical methods utilized are (1) removal of certain sympathetic ganglia; (2) ablation of some of the vascular plexuses of the sympathetic; (3) occlusion of the middle meningeal artery.

G. D. Dickerson[6] reports some very encouraging results with the third method enumerated above. In five of six cases typical of migraine, the sixth being somewhat questionable, either cure or definite relief has been produced for varying periods of time.

Riley points out, however, that a final decision as to the permanency of such results should not be reached at the present time, for it is well known that almost any form of radical change in physiology, metabolism or even psychic attitude in a patient may result in a cessation or abatement of migrainous attacks.

THE MENINGES AND CEREBROSPINAL FLUID

In a detailed paper, F. Fremont-Smith[7] gives the causes of the alterations in the cerebrospinal fluid in meningitis. The increase in pressure is due to several causes, the most important of which are (1) dilatation of the intracranial blood vessels, and (2) mechanical obstruction by the exudate and the inflammatory reaction of the cerebrospinal fluid pathways. Decrease in the osmotic pressure of the blood, increase in the osmotic pressure of the cerebrospinal fluid and edema of the brain may also play a rôle. The cellular exudate comes chiefly from the blood stream, and to a lesser extent from the arachnoid cells; it is the usual cellular response to infection.

In meningitis, as in health, the chemical composition of the cerebrospinal fluid depends chiefly on the composition of the blood plasma, the cerebrospinal fluid tending to remain in osmotic equilibrium with the blood. The chief change in composition of the blood plasma in meningitis is a lowering of the chlorides, which is reflected by a parallel fall in cerebrospinal fluid chlorides. A similar fall in cerebrospinal fluid chloride occurs whenever the plasma chlorides are decreased, notably in acute febrile diseases, such as pneumonia, scarlet fever, etc.

Another factor influencing the composition of the cerebrospinal fluid in meningitis is the local breakdown of dextrose as a result of bacterial and cellular action. This results in a lowering of the dextrose content of the cerebrospinal fluid, which may fall nearly to zero and in an increase in acidity of the cerebrospinal fluid, chiefly due to an increase in lactic acid. This, in turn, results in a disturbance of the Donnan membrane-equilibrium and tends to lower the chloride content of the cerebrospinal fluid, bringing it nearer to that of the blood.

In addition, there is a partial breakdown of the impermeability to protein of the membranes separating the plasma from the cerebrospinal fluid. This allows protein to enter the cerebrospinal fluid, raising its protein content, which results in lowering of the cerebrospinal fluid chlorides. As the protein content of the cerebrospinal fluid in meningitis is usually only moderately increased, so that when compared with plasma, the cerebrospinal fluid remains relatively protein-poor, the effect of this increased permeability on the chloride distribution is usually slight and often negligible. Occasionally, however, especially in the presence of subarachnoid block, the protein content becomes markedly increased. Under these circumstances, there is also the opportunity for the greatest accumulation of lactic acid. The cerebrospinal fluid chlorides level may then become appreciably closer to that of the plasma. Rarely this combined effect may be quantitatively greater in lowering the cerebrospinal fluid chlorides than the effect of the fall in plasma chlorides.

EFFECT OF SPINAL ANESTHESIA ON CENTRAL NERVOUS SYSTEM

F. G. Lindemulder[8], in a review of the literature and from his own personal observations, points out the sequelae and danger of this method of anesthesia. He found that the most common sequel was pain in the extremities, especially in the legs, but also in the back and, in one instance, the head. In three cases, the pain in the legs continued for several months. Marked muscle tenderness in the extremities also resulted in these three cases.

In one case, in which a sensory level reached the second cervical segments, the patient showed the following: severe headache, neck rigidity, positive Kernig, active tendon reflexes, marked hyperesthesia over the body, Babinski on the left, loss of vibration sense over the ankles, and a loss of the sense of motion and position of the toes. Within 24 hours there was noted a paralysis of the right external rectus muscle. These symptoms lasted for several weeks, and except for the pains in the arms and legs, the patient was asymptomatic at the time of his discharge from the hospital. The spinal fluid was negative after the operation.

In two patients that died several days following the operation, the spinal anesthesia is thought to have been a contributing cause. Necropsy showed in both cases severe urinary tract infection, congestion, edema and degenera-

tions in the brain and cord, attributable to the anesthesia.

The author advances the theory that all spinal anesthesias produce an acute myelitis which usually lasts a few hours and may leave the patient with complaints of pain and paralyses for a period, but at times there may be a permanent involvement of the nervous system, both clinically and pathologically. That spinal anesthesia may cause definite organic changes in the cord and membrane is pointed out by Loyal Davis, Hale Haven, J. H. Givens and John Emmett[9] who injected the common solutions in use in the dural sacs of dogs. They found (1) a varying degree of inflammatory reaction in the leptomeninges; (2) passive changes in the ganglion cells of the grey matter of the cord, similar to those seen in so-called wallerian degeneration; (3) swelling and fragmentation of the axis-cylinders; (4) signs of degenerative changes in the fibre tracts of the cord. Although the first three of these changes were not pronounced and were usually seen to disappear gradually, the inflammatory changes in the lepto-meninges were constantly present.

TRAUMATIC AFFECTIONS

S. Bernard Wortis[10] observed the following changes after producing experimental cerebral lacerations in cats: (1) aseptic lacerations of the brain without removal of the products of trauma in the cat results in ventricular distortion due to (a) meningocerebral adhesions and (b) contracting cerebral cicatrix; (2) head trauma resulting in the escape of blood into the cerebrospinal fluid often gives rise to mild bilateral ventricular dilatation in the absence of grossly demonstrable meningocerebral adhesions or a cerebral scar; (3) aseptic laceration of the brain and head trauma resulting in fracture of the skull, increase the animal's sensitiveness to a standard convulsant over the period of observation.

Mechanisms in Brain Trauma

According to his clinical observations, N. W. Winkleman[11] states that blood in the subarachnoid space acts as an irritant and produces the clinical picture of a meningeal inflammation and pathologically the so-called aseptic meningitis. Because of this, the author advises frequent spinal drainage to prevent such a reaction in the meninges. Spinal drainage in such cases serves to directly relieve the symptoms.

Subarachnoid bleeding may secondarily result in a blockage to the spinal fluid absorption through the pacchionian filters and a damming back of the fluid over the vertex. If this continues, atrophy of the convolutions will result. (The present authors regard these views as somewhat extreme, although agreeing that lumbar puncture in moderation is valuable.)

Winkleman advises caution in making the diagnosis of posttraumatic neurosis, even in the absence of all clinical signs of involvement of the brain. Many so-called causes of neurosis, following head trauma, may be shown to have an organic basis by encephalographic studies.

Encephalographic Observations on Head Injuries

E. D. Friedman[12] reports that in a group of head cases, showing either focal or generalized cerebral symptoms, all but one showed definite changes in the encephalogram: dilatation of the ventricles, considerable accumulation of air on the convexity of the brain and migration of the ventricular system toward the site of the lesion. These deviations from the normal were found either separately or in combination. Friedman states that these encephalographic observations were so uniformly abnormal that they are suggestive of an organic basis for some of the symptoms in the posttraumatic state, not only in those cases in which focal signs are present, but also in those cases in which general symptoms are complained of. Encephalography thus offers a valuable means of differentiating organic and functional syndromes.

Evaluation of Evidence in Head Injury Cases

Foster Kennedy[13] offers the following criteria of head injury sufficient to produce organic changes in the brain:

A. Absolute criteria:
   (1) Roentgen evidence—skull fracture.
   (2) Bloody spinal fluid.
   (3) Bleeding from the orifices, especially the ears.
   (4) Focal cerebral palsies.

B. Presumptive criteria in the order of their importance:
   (5) Convulsive states proved to be posttraumatic.
   (6) Ventricular distortion, proved to be posttraumatic.
   (7) History of prolonged unconsciousness.
   (8) History of adequate trauma with especial consideration to the occurrence of vomiting following the injury.

If headache and dizziness persist for more than four months in a man under 60 unassociated with the first seven signs listed above, they are to be regarded as "suggested neuroses" unfounded in structural change.

Head Injuries and their Complications

Most cases of head injury, Gilbert Horrax[14] finds, fall into the following groups: (1), those spoken of as "concussion," in which loss of consciousness is brief and with or without fracture of the skull; (2), those in which there is

considerably more intracranial damage than is ordinarily implied by the term concussion and usually a demonstrable linear fracture of either the base or the vault; (3), those with profound unconsciousness or even moribund; (4) those who present the typical syndrome of an extradural clot from meningeal hemorrhage; (5), those with depressed fractures; and (6), those with gunshot wounds. The author discusses the treatment of each group and the complications of head injury, including meningitis, abscess, subdural hematoma, and pneumocephalus.

Subdural Hematoma

Papers on this subject by Abraham Kaplan[16], H. W. Fleming and O. W. Jones, Jr.,[16] and W. J. Gardner[17] point out the following salient features:

1. The condition is frequently unrecognized, although more common than middle meningeal hemorrhage and frequently follows insignificant head injuries.

2. The interval between the injury and onset of symptoms varies from a few days to many months.

3. There is no typical clinical picture. Misleading and bizarre pyramidal tract signs may be noted, such as ipsolateral hemiplegia. According to Kaplan, the most characteristic symptom of chronic subdural hematoma is a peculiar lethargic state which seems to alternate from drowsiness to coma, with periods of alertness and surprising response occurring within 24 hours or several days. Early symptoms, state Fleming and Jones, often simulate those ascribed to posttraumatic psychoses; and when followed by intracranial signs of general and local pressure or irritative phenomena are most significant. The spinal fluid pressure is usually definitely but moderately increased and, as a rule, is clear; occasionally, it is xanthochromic.

4. Operation, if done early enough, will usually be followed by recovery.

VASCULAR AFFECTIONS

Spontaneous Subarachnoid Hemorrhage

I. Strauss, J. H. Globus and S. W. Ginsburg[18] give a long and well-illustrated report of 34 cases, describing this syndrome. These authors point out that it is a disease entity that should be clearly understood, not only by the neurologist, but by the interne who is more likely to see the condition in the acute stage.

The syndrome is characterized by a short prodromal stage, during which headache, dizziness, nausea, and rigidity of the neck and spine are the outstanding symptoms. This is followed by an abrupt and explosive onset of the acute stage with violent headache, impaired consciousness and occasionally by convulsions. The usual signs of meningeal irritation and of increased intracranial pressure are found, the cerebrospinal fluid now being usually bloody. The clinical course may be short or last several weeks, with or without recurrences of bleeding.

It is important to differentiate this type of bleeding and other forms of intracranial hemorrhage for, once recognized, the treatment is very definite: repeated lumbar punctures to relieve the symptoms of increased intracranial pressure. The prognosis, though always grave, is not hopeless; recoveries are frequent.

Brain Changes in Malignant Endocarditis

I. B. Diamond[19] found from the examination of 12 brains from cases of malignant endocarditis that changes in this organ are frequent. The lesions may be either circumscribed or diffuse, the former usually being in the form of nodules, the latter in the form of meningoencephalitis. These changes are manifestations of a defense reaction against an infection or intoxication.

Spinal Fluid in Arterial Hypertension

Samuel A. Shelburne, Daniel Blain and James P. O'Hare[20] made a study of 50 patients showing arterial hypertension. In 21 patients there was increased intracranial pressure, nearly all of them having edema of the optic discs. Although it was more common to find renal disease associated with these changes, they were also found in the presence of normal renal function. The authors could find no cause for the increased intracranial pressure in 50 per cent of the cases.

Francis C. Grant[21] describes five cases with the syndrome of malignant hypertension characterized by headache, vomiting, and choked discs. In two cases there was such definite neurological findings that a diagnosis of neoplasm was made. Operation, however, failed to reveal a tumor. In none of the cases was there sufficient evidence of renal disease to account for the hypertension.

The Encephalitides

The pathogenesis and the classification of disseminated encephalitides and the demyelinizing diseases are still in a state of great confusion. Thus, Rivers in an article on filterable viruses quotes several workers who consider such diseases as multiple sclerosis, as a degenerative rather than inflammatory condition, while other investigators consider them as due either to single or combined infections. Levaditi and Pette insist that all the demyelinizing diseases are caused by filterable viruses.

Such differences of opinion as to etiology together with difficulties of classification may be seen by reading papers on this subject by Rivers[22], Gill and Richter[23], Bielschowsky and Maas[24], Barrera[25] and O. Sager and D. Gregoresar[26].

Charles F. McKhann[27] gives an instructive picture of lead encephalitis in children. This author states that lead produces severe cerebral involvement, probably due to marked edema,

which may, if the children survive, leave permanent neurological disorders. Peripheral neuritis is less common in children than in adults. In the acute stage, treatment must be directed toward the relief of the greatly increased intracranial tension.

Encephalitis from Stovarsol

According to Reiter[28], stovarsol, an arsenical compound, used in the treatment of Vincent's angina, may cause a severe dermatitis, fatal agranulocytosis, and hemorrhagic encephalitis. Reiter describes a case of a man who received 55 grams of the drug in seven days. He developed a rash, convulsions, left facial paralysis, loss of knee jerks, athetoid movements, and stupor. He was given intravenous glucose and insulin, and recovered.

NEUROSYPHILIS

Histopathology of Therapeutic Malaria

In a very detailed histologic study of the tissue changes in a syphilitic (not paretic) patient who died while under treatment for malaria, Walter L. Breutsch[29] concludes that one of the many factors accounting for the beneficial effects of therapeutic malaria is the activation of the reticulo-endothelial system accompanied by new formation of macrophagic tissue. In the nervous system, the reticulocytic response is greatest in the leptomeninges and to a lesser extent in the perivascular spaces of the large vessels, in the white matter in the striatum and in the pons. Activation of the reticulo-endothelial system elsewhere in the body occurs in addition to an increased response in the undifferentiated embryonic mesenchymal cells. (Abnormal cells belonging to the reticulo-endothelial system have been found in the blood of general paretics not treated with malaria by Dr. William Dameshek working in the laboratory of the Boston State Hospital.)

The Treatment of Neurosyphilis

H. C. Solomon[30] makes some very pointed and valuable statements as to the management of neurosyphilis. He stresses the great importance of considering every case of syphilis a potential prospect for the development of neurosyphilis. This calls for a careful neurological survey of the infected patient from the earliest to the latest period of the disease. Once there is evidence of central nervous system involvement, the emphasis must be laid on the type of therapy that will produce serological improvement. If in the early stages of the disease, arsphenamine, bismuth, and mercury have been found to be ineffective in producing a serological cure, one must turn to either tryparsamide or fever therapy, or a combination of the two. If this is adhered to, there is great likelihood that the central nervous system involvement may be eradicated, or at least held in check.

Although tryparsamide and malaria are considered to be the most beneficial remedies in general paresis, there is a difference of opinion among neurologists as to the order in which each type of therapy be given. According to W. A. Caldwell[31], the most effective method is a short course of tryparsamide before the induction of the pyrexia. In this way, there is a decrease in the cortical spirochetosis and an improved general condition, so that the patient is able to withstand the exhaustion of the fever.

Sulphur in General Paresis

Injections of sulfosin give as good results in this type of neurosyphilis as does malaria according to experience in Denmark for the past five years. Knud Schroeder[32], who reports the results of the treatment, states that sulphur in oil gives a maximal therapeutic effect with minimal risks to the patient. It is, however, too soon to compare the durability of its good effects with those of malarial treatment.

THE SPINAL CORD

Spinal Epidural Abscess

Several cases of abscesses of the epidural space have been reported this past year. A. Bellerose and R. Amyot[33] relate the case of a woman, 39 years old, who was being treated for multiple furunculosis of the back of her neck. She later developed a chill, fever, and intercostal pain. Several days later, she had difficulty in urination, followed in two days by signs of transverse myelitis. Lumbar puncture showed xanthochromic fluid which coagulated spontaneously. Laminectomy showed a collection of thick yellow pus in the epidural space at the fifth thoracic vertebra. This was evacuated but the paraplegia persisted. The patient later developed new abscesses and died.

S. S. Allen and E. A. Kahn[34] report three cases of epidural abscess. In the first instance, following recovery from appendicitis, subphrenic abscess and septicemia, a man developed signs of a myelitis with signs of block at the third thoracic vertebra. In the second case, a girl developed epidural suppuration at the thoracic region. This occurred following a fall. Death occurred in both cases, although surgery was done. In the third case, the abscess developed in a man with a furuncle on the back of his neck. Laminectomy disclosed at the level of the seventh thoracic segment a large amount of pus which was evacuated. The patient survived, although he remained paralytic.

W. J. Mixter and R. H. Smithwick[35] report ten cases of acute spinal epidural abscess, following other infections, in five of which vertebral osteomyelitis was associated with the suppuration. Of eight who had laminectomies, three recovered.

MULTIPLE SCLEROSIS

Etiological Factors

J. S. Collier[36] discusses the household incidence as opposed to the familial incidence of this disease. Because he had noted on three different occasions that two young male patients entirely unrelated, but who had been brought up in the same environment developed the ailment, he suggests a common infection in the three pairs. On the other hand, all cases cannot be explained in this way, for the author has also observed the condition in a mother and two daughters. The mother died when the daughters were infants, and furthermore, for 15 years prior to the onset of the symptoms in the younger one, the girls had not seen each other. This, of course, argues for a familial incidence.

Another possible etiological factor in multiple sclerosis has been suggested by R. S. Allison[37]. He has observed from cases occurring in Ireland that the only frequent feature in the large majority of cases was the fact that they obtained their drinking water from spring wells. There also seemed to be a relationship between multiple sclerosis and goitre because of similar geographical distribution of the two diseases; possibly the link between the two diseases is drinking water. (Both these theories are interesting but to the reviewers seem hardly convincing.)

Therapy in Multiple Sclerosis

Not only does fever therapy exaggerate the symptoms of this disease, but, states J. C. Mussio Fournier[38], injections of typhoid vaccine cause the appearance of added symptoms, such as ptosis, diplopia, hyperalgesia, ankle clonus, and diminished acuity of vision. The author suggests because of these observations that vaccine injections may settle a diagnosis in early cases, in which there are no objective signs and it is impossible to differentiate disseminated sclerosis and hysteria.

Richard M. Brickner[39] reports favorable results in 40 cases of multiple sclerosis with quinine. The usual dose given was five grains three times a day. In the majority of instances, improvement was noted in those symptoms which had been present for not more than two years. The author points out that relapses should not cause the treatment to be abandoned, since in no case did the relapse last longer than from four to six weeks when treatment was continued. The quinine should be continued indefinitely to maintain relief of symptoms. If cinchonism develops the treatment should be temporarily interrupted and then quinine given in smaller doses. Many symptoms were noted to regress, particularly spasticity.

PERNICIOUS ANEMIA

Neurological Manifestations

In a series of 515 cases observed over a period of 18 years at the Peter Bent Brigham Hospital, Richard H. Young[40] reports the following neurological features of pernicious anemia: Twenty per cent of the cases showed marked cord changes as exhibited by reflex changes and ataxia. Four and five-tenths per cent showed psychotic trends of varying nature, including acute deliriums, depressions, paranoid states, and memory and disposition changes. Changes in disposition were often associated with the progress of remissions. Autopsies in the cases showing psychotic changes revealed several pathological alterations, including a chronic pachymeningitis and leptomeningitis in one case, and a subdural clot in another case. Other neurological features presented were nystagmus, scanning or staccato speech, dysarthria associated with aphasia, a sixth nerve palsy, and two cases of seventh nerve palsy.

Liver Therapy

From their experience with the effect of liver therapy on the neurological manifestations of pernicious anemia, Benjamin M. Baker, Jr., James Bordley, III, and Warfield T. Longcope[41] point out the importance of the observation stressed by other investigators, that before expecting marked improvement, large amounts of liver must be administered over long periods. Thus, in cases treated for less than six months, improvement occurred in 31.25 per cent of the neurological signs and symptoms; in those treated for more than six months, 55.17 per cent of the signs were improved, and in those treated over ten months, 58.93 per cent of the signs of combined degeneration were favorably influenced.

The authors discuss the possible implication of vitamin deficiency in the production of some of the symptoms and signs which may be dependent upon changes in the peripheral nerves.

THE PERIPHERAL NERVES

Peripheral Paralyses Following Serum Treatment

Several new cases of peripheral nerve involvement following injections of serum and antitoxin continue to be reported.

Forest Young[42] reports the case of a man, aged 40, who developed serum sickness three days after an injection of prophylactic tetanus antitoxin. Three days later he complained of pain in the arms, in the back, and legs. Seven days after the onset of the serum sickness, he developed weakness of the right arm with diminished sensibility. Five weeks later the axillary nerve was paralyzed and showed a complete reaction of degeneration; atrophy of the

deltoid was also present. Only slight motion of the deltoid returned after three months of massage and abduction in a splint.

George Wilson and Samuel B. Hadden[43] report six cases of involvement of the circumflex nerve, with slighter involvement of other nerves, following the injection in four patients of tetanus antitoxin and of diphtheria antitoxin in two cases. Following inoculation against scarlet fever, Alfred Gordon[44] observed three cases of musculo-spiral paralysis and one case of facial palsy. These patients all recovered in from four to seven weeks.

Arsenic and Lead Neuritis

After making chemical studies in 33 cases of neuritis in which lead or arsenic was found, Walter D. Shelden, John B. Doyle and Arnold E. Osterberg[45] make some very interesting observations. They find that lead is retained in the body much longer than arsenic. The amounts of these substances bear no particular relationship to the clinical condition. Patients with large amounts of arsenic in the urine may exhibit no signs of arsenical poisoning and may indicate only that the intake has been recent. On the other hand, the absence of the metal from the urine and its presence in the hair may indicate that it has been stored up for a long time and yet cause toxic symptoms. The same may hold in cases of lead poisoning. The authors cite cases exemplifying the above phenomena.

Multiple Neuritis from Apiol

A multiple neuritis resembling Jamaica ginger neuritis has been found to result from the administration of apiol, a substance used as an abortifacient. According to Karl Hellmuth and Richard Grün[46], the toxic effect is not due to the apiol itself but to admixture of triorthocresolphosphoric ester. This substance is also the cause of Jamaica ginger paralysis. The above authors report a case of multiple neuritis resulting from ingestion of large amounts of apiol to induce abortion.

Emil Rechnitz[47] reports six cases of apiol multiple neuritis occurring in Jugoslavia. The symptoms in each case were alike, first paresthesia, then paralysis of hands and feet. Usually the knee jerks were preserved, the ankle jerks lost; sensation remained intact.

Another case is reported by Henry Roger[48]. In this instance, that of a young woman, all four extremities were paralyzed. A severe case of multiple neuritis is reported by H. Jagdhold[49]. The patient, a woman of 31, had taken nine capsules of apiol for five days for the purpose of inducing abortion.

Etiology of Polyneuritis

I. S. Wechsler[50] points out the importance of food deficiencies in the production of polyneuritis. From his observations of clinical material, he concludes that many cases of obscure origin are probably neither toxic nor infectious in nature, but more likely are deficiency syndromes, that is, belonging to the group of avitaminosis. Thus, one can often elicit a history of prolonged loss of appetite, diarrhea or vomiting, absence of free hydrochloric acid, or evidence of gastro-intestinal or hepatic disease.

In many cases of polyneuritis, hitherto regarded as caused solely by a specific factor, such as alcohol or the metals, one often finds an additional factor of avitaminosis which may possibly be of more importance than the so-called specific cause. This view may explain why the polyneuritis develops in certain of the patients although all are exposed to the same toxin. In any case, one should apply the therapeutic test of giving diet rich in vitamines even though a specific cause may not be found. Although as yet, there is no evidence pointing to which vitamin is involved in the obscure cases or in cases in which avitaminosis seems to play an important part, there is some experimental evidence that absence of vitamin A and possibly also of C and D can lead to degenerative changes in the spinal cord, the roots and the nerves, and furthermore, that their presence will prevent degeneration by toxins which sometimes affect the nervous system.

The Vegetative Nervous System

J. F. Fulton[51], the physiologist, gives an excellent résumé of our present knowledge of the hypothalamic region. Not only does the hypothalamus influence the heart and intestinal tract, but it regulates carbohydrate, fat, and water metabolism. Furthermore, heat regulation, vasomotor control, sexual function, and the activity of all other organs innervated by the autonomic nervous system are, to some extent, controlled by this area. The real significance of this intimate relationship with so many bodily functions has been pointed out by Cannon. The sympathetic centre of the autonomic system, located in the posterior hypothalamus, brings about those adjustments essential for escape and combat, or what may be called adjustments of a spendthrift character: elevation of the temperature, acceleration of the heart, and mobilization of the energy reserves. On the other hand, activity of the parasympathetic division, calls forth a defense mechanism of conservative character. Here the temperature falls; the pupils contract; the glycogen reserves of the body become replenished; and gastro-intestinal activity is increased; and foodstuffs are made available to the organism.

The author points out that it is evident more and more that the remarkable constancy of the internal milieu of the body, temperature, salt, hydrogen-ion concentration, etc., is controlled mainly if not entirely by the various centres of

the autonomic nervous system. This conception opens new horizons to the physiologist, for it allows him to visualize more adequately than ever before the integrated functions of the organism as a whole.

Muscular Dystrophy and Myasthenia Gravis

Interesting chemical studies with respect to creatin have been made and therapeutic effects of glycine in these two conditions have been observed by several workers. A. T. Milhorat, F. Techner and K. Thomas[52] have followed the course of three cases of progressive muscular dystrophy and three cases of pseudo-hypertrophic muscular dystrophy, in which the daily ingestion of glycine caused a definite increase in the excretion of creatin. After several weeks a definite clinical improvement associated with a decrease in the excretion of creatin occurred.

W. M. Boothby[53] finds that even on a creatin-free diet, patients with progressive muscular dystrophy continue to excrete creatin. Four patients, who were put on a normal diet, showed creatin nitrogen equal to, or in excess of, the preformed creatinin nitrogen.

Patients with myasthenia gravis, on the other hand, excrete little or no creatin. The author reports six cases of myasthenia gravis, which were benefited by glycine. One of the patients, who previous to treatment could walk only two blocks and had such marked weakness of his arms and shoulders that he could hardly shave, marked ptosis of the eyelids and difficulty in swallowing, showed improvement of these symptoms after taking 15 grams of glycine twice daily for several weeks. Another patient made a startling improvement also. A third made some improvement. Therapy on the other three patients had to be discontinued because of an insufficient supply of glycine.

L. Remen[54] has observed two cases of myasthenia gravis, which were given glycine in doses of ten grams for two months. Definite improvement occurred. During treatment, the creatin, which had been considerable, disappeared from the urine.

The only treatment of any value for muscular dystrophy, according to G. Goralewski and E. Engel[55], is hypodermic injections on alternating days of adrenalin and pilocarpin of each .2 cc. of a one per cent solution. They give the report of one case that was given 50 injections. Careful measurements of the volume and the strength of certain muscles were made before and after treatment. (The evidence adduced is not convincing.)

Muscular improvement was observed by Anna Leiter[56] in three cases of dystrophy that were given 0.03 cc. adrenalin of 1/1000 solution intravenously, the dose gradually being increased to .15 cc. Thirty doses were given daily or every other day. The blood showed a rise in the previously low sugar content. (The reviewers' comment is as above.)

Etiology of Peptic Ulcer

The implication of neurogenic factors in the etiology of peptic ulcer is stressed by Harvey Cushing[57]. He cites three cases in which perforation of acute ulcers of respectively the stomach, duodenum, and esophagus occurred soon after operation for cerebellar tumor. Cushing also discusses two cases of chronic duodenal ulcer, in one case of which cerebellar tumor was present, and in the other a tumor of the third ventricle was found. He cites also two cases of mucosal erosions associated with cerebellar tumor and olfactory groove meningioma respectively. Cushing, in pointing out the psychic aspect of the ulcer problem, states that it is common knowledge that "highly strung" persons are particularly susceptible to nervous indigestion with associated ulcer; that mental and physical rest has a definite beneficial effect on the course of the ulcer and often tends to heal them; furthermore, symptoms often are prone to recur when the patient returns to his former habits.

In attempting to find a reasonable explanation for the etiology of ulcer, Cushing cites the fact that experimental lesions made anywhere along the course of the fibre tracts from the anterior hypothalamus to the vagal centre are prone to cause gastric erosions. In the light of such evidence, it may well be that the interbrain, now recognized as a highly important centre, may be so adversely affected by psychic influences arising in the so-called vagotonic individual, as to finally result in the production of ulceration of the upper alimentary canal.

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MOTHER'S DAY

Mother's Day, May 13th, is to be observed by women's clubs, men's clubs, medical societies, chambers of commerce and other professional and civic groups who will join in community efforts throughout the nation to "Make Motherhood Safe for Mothers." This announcement is made by Mrs. Shepard Krech, President of the Maternity Centre Association, New York City.

"An important step forward is to be taken this year," states Mrs. Krech. "In three previous Mother's Day Campaigns, the effort was to arouse the public to the importance of the fact that two-thirds of the maternity deaths are preventable; that 10,000 of the 16,000 women who annually die in childbirth could be saved. The next move, which is to be taken this year, is to do something quite specific and definite about it, with groups working in every community."

Mrs. Krech stated that specific changes cannot be made to improve conditions until people study their own local maternity facilities and determine just what is needed. She indicated that the phases requiring attention may differ widely in various communities.

"Blank appraisal forms," added Mrs. Krech, "are available. By the use of these, any group of persons may conduct an investigation into the adequacy of what their own town or county is doing for mothers. Such questions as these are to be answered: 'Number of maternity beds?'—'Total number of births in the last year?'—'Number of deaths in the last year?'—'Is organized prenatal nursing service provided?'—'Is your hospital approved by the American College of Surgeons?'

"There has been a great deal of sentiment surrounding Mother's Day during the time that it has been observed in this country," said Mrs. Krech. "In the last few years there has been a successful effort to direct this fine feeling toward the important subject of saving mothers from unnecessary death. But emotion alone is not enough. We must have facts, pertinent facts, local facts, so that groups in every community may work with their own physicians, health officers, nursing associations and hospital authorities to alter those factors in the situation which are a barrier to safe motherhood.

"Only by an appraisal of maternity facilities in every community, and study of their quality, can the great step forward be taken. These blanks are available free. Prizes are to be awarded those groups which have the highest rating for making a thorough-going survey and presenting a plan for improvement based upon that survey. Programs for club meetings are also available without charge, as well as publicity material for local Mother's Day Campaigns designed to direct the sentiments surrounding this occasion into channels that will be productive of results in terms of human lives saved."

The Maternity Centre Association is a voluntary organization supported by private contribution. The address is 1 East 57th Street, New York City.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 20061

PRESENTATION OF CASE

A retired American banker seventy years old entered complaining of vomiting of two weeks' duration.

The patient was first seen as a private patient of Dr. Wyman Richardson's about eighteen months before admission. At that time he complained of lower abdominal pain and gas of three months' duration. This pain was associated with a severe mid-dorsal pain and produced a steady discomfort. The pain was often brought on by exertion. He occasionally complained of heartburn. His bowels were regular. He had lost five pounds during the past six months. There had been no bloody or tarry stools. Physical examination was entirely negative except for a slightly distended abdomen and a little resistance in the left lower quadrant. There were bilateral inguinal herniae. A gastro-intestinal series showed a fleck ulcer of the duodenum without evidence of obstruction or appreciable deformity. The esophagus was normal. He was put on a six-meal bland diet and tincture of belladonna ten drops three times a day. His pain disappeared for a while, but recurred with dietary indiscretions. Five months before admission he was having a great deal of pain. He was very much depressed over his financial situation. He developed slight edema of the ankles. He was warned to keep on his diet.

Two weeks before admission he suddenly noticed a sharp pain under the sternum associated with discomfort upon swallowing. The pain remained localized under the sternum and did not radiate. It became so severe that he was afraid to swallow, although he had been on a liquid diet for the past year. Shortly after the onset of pain he began to vomit after one or two swallows of liquid food. The vomitus or probably the regurgitated food was frothy but never curdled. The vomiting occurred sometimes immediately after swallowing and at other times as long as five to ten minutes later. The pain on swallowing and the vomiting persisted to the day of admission. There was no history of hematemesis or dysphagia at any other time previous to this. His bowel habits were normal. There were no bloody or tarry stools. During the past six months he had lost strength and about ten or twelve pounds in weight.

His marital, family and past histories are irrelevant.

Physical examination showed a thin, somewhat pale old man with dry, wrinkled skin and evidence of dehydration and weight loss. The teeth were false. There was a moderate dorsal kyphosis. His chest was hyperresonant to percussion. The expansion was somewhat limited. The heart sounds were very faintly audible. The blood pressure was 120/80. There were bilateral inguinal herniae.

Examination of the urine was negative except for a few hyaline casts. The blood showed a red cell count of 5,050,000 with a hemoglobin of 85 per cent, a white cell count of 6,900, and 63 per cent polymorphonuclears. The stools were negative.

A gastro-intestinal series showed almost complete obstruction in the esophagus at the level of the ninth dorsal vertebra. The aorta crossed the midline at the site of the obstruction in the esophagus but did not appear dilated. There was also probable calcification in the aortic cusps without hypertrophy of the heart. During the first five or ten minutes of the observation no barium passed through the point of obstruction. Later however when the patient had drunk as much water as possible a thin stream of barium passed the point of obstruction. The esophagus was quite smooth and conical down to the point of obstruction. There was no evidence of tumor growing into the esophagus or ulceration at the site of stricture. There was an orange-sized herniation of the stomach through the diaphragm.

Two days after admission an adult sized esophagoscope was passed about twelve inches and almost complete stenosis of the esophagus was encountered. Not even a small bronchial bougie could be passed through this stricture. The posterior esophageal wall just above the stricture bled very easily but no outeropping could be seen.

The following day a Witzel gastrostomy was performed under local anesthesia. The next morning the patient was disoriented. He became comatose during the day. Numerous tracheal râles were heard throughout both lungs. He died soon afterwards.

CLINICAL DISCUSSION

DR. TRACY B. MALLORY: Dr. Richardson, have you anything to add to the history?

DR. WYMAN RICHARDSON: I think it is pretty accurate from my point of view. I would say this. He was a very difficult patient to get an accurate history from. His symptoms would vary from visit to visit, depending a good deal on how he happened to feel on that particular day. I do not think there is anything else that I can add to the history.

DR. AUBREY O. HAMPTON: When this man came in the first time I found a duodenal ulcer and apparently did not look at this film at all, because here is a perfectly obvious hernia of the stomach through the diaphragm. I probably dictated the notes from the fluoroscopic examination alone, and the hernia may not have been present during that examination. I do not know whether that is the duodenal ulcer or not; I think it is. He had nothing at that examination that would indicate any delay in his esophagus. He drank the usual quantity of barium rapidly and I did not see any reason to suspect the lesion in the esophagus.

At this examination, fifteen months later, he had almost complete obstruction of the esophagus. This lesion was undoubtedly in the region of the cardiac sphincter. Here is a much better filling of the portions of the stomach above the diaphragm. We thought that the lesion was too smooth and conical for an ordinary carcinoma, and since it was in the region of the sphincter an ulcer or cardiospasm would produce the picture.

Since he had an ulcer of the duodenum I think we spent most of our time trying to find an ulcer in the esophagus, but we could not find it. This comma-like shadow in the region of the sphincter of the esophagus is supposed to be normal.

We did see some calcification in and around the heart and aorta. That interested us, but I was afraid to write it in the record. I think it pulsated like an aortic valve, but I could not see it very plainly and there were other shadows around it, so I did not have the courage to put it into the record.

DR. EDWARD D. CHURCHILL: Do you call this a thoracic stomach, or a diaphragmatic hernia?

DR. HAMPTON: I should say it is an ordinary diaphragmatic hernia of the stomach.

DR. CHURCHILL: With a short esophagus?

DR. HAMPTON: When you diagnose a short esophagus you are dealing with something that is very rare. In this case we have been trying to explain the shortening of the esophagus as contracture secondary to ulcer. I do not know whether that is too theoretical or not. We know that the cardiac orifice of the diaphragm is often large and relaxed in elderly people.

DR. MALLORY: I will ask Dr. Churchill to discuss this case.

DR. CHURCHILL: This combination of hernia through the esophageal orifice plus the obstruction in the cardiac end of the stomach is a new one to me. It brings up a point upon which a good many radiologists seem to be in disagreement, whether most of the so-called hernias through the esophageal orifice are due to a short esophagus and the condition should be called thoracic stomach, or whether the lower end of the esophagus is in the normal place within the cardiac end of the stomach expanding upward through the diaphragm.

Of course, speaking on the basis of statistics, esophageal obstruction of less than eighteen months' duration coming out of a clear sky in a man of seventy means carcinoma. That diagnosis must be held in favor despite the negative evidence from esophagoscopy and the information of the X-Ray Department to the contrary. However, with the short esophagus it suggests that the possibility of ulcer might be very real, and we know that herniation of the stomach through the diaphragm is associated with erosions of the mucosa. Dr. Bock has shown it to be the cause of constant loss of blood in the stools leading to secondary anemia. There was no secondary anemia in this case, I take it. There is no positive guaiac recorded in the stool examination, so we have no evidence that he is losing any blood. The differential diagnosis must rest between an ulcer in the region of the cardia involving the stomach and esophagus with a chronic strieture, and a carcinoma. Sticking to our numerical law of chances I should favor carcinoma, despite the evidence to the contrary.

I may say that this man seems especially constructed for an operation on the lower end of the esophagus. What we have been trying to do is to resect the carcinoma and bring the stomach up into the retropleural space, but in this case it is already up there so resection might be a relatively simple thing.

Why he died I do not know. We have not enough data. I think the gastrostomy might have been delayed a little. Esophagoscopy one day and gastrostomy the next is a strenuous sequence for a man of seventy in an emaciated condition. I think these patients should be prepared with intravenous glucose and intravenous saline, and the less done at one time the better. Perhaps the gastrostomy might well have been done before the esophagoscopy. I should not like to do a gastrostomy the day after an esophagoscopy because if the patient died I might get the blame when it really should rest with my colleague who did the esophagoscopy.

DR. ARLIE V. BOCK: I think the evidence in the literature is such that a short esophagus would be a relatively rare finding in cases with hernia of this type. Neither of the two cases on which we have had complete postmortem examinations had such an esophagus. I believe the esophagus is of normal length in most cases. I do not believe that the strieture mentioned has any relation to the hernia.

DR. HAMPTON: The reason I brought that up was because of the patient Dr. Young has. This patient has stricture due to lye ingested at the age of two. He has a very short esophagus and a hernia similar to this one. I do not know anything about it. That is just a theory.

DR. GEORGE W. HOLMES: I would agree with Dr. Bock that the true short esophagus is a con-

genital lesion and not likely to be acquired. These herniae through the esophageal meatus occur in people past middle life. They are quite common. It does not seem at all likely that the length of the esophagus has anything to do with it.

DR. MALLORY: What do you think about it, Dr. Mosher?

DR. HARRIS P. MOSHER: My first guess as to the pathologic condition would be carcinoma, my second ulcer.

As for the short esophagus, I see very few of them. The cases all come over here, but the literature of late—I have in mind the *British Journal of Laryngology*—shows that the short esophagus is not uncommon in children. The article I refer to reported five or six cases. I have always been in doubt about biopsies of the mucous membrane of the esophagus as being sure proof of misplaced gastric mucosa.

DR. MALLORY: Do you know anything about that, Dr. Bremer?

DR. J. L. BREMER: No, but it occurs to me that you are stating that a certain portion is a stomach because it has gastric mucosa. We do not know at all what makes gastric mucosa grow in one part, esophagus grow in another part of the tube, and intestine in another. I do not know why we speak of the tube as esophagus or stomach merely on account of the type of mucosa. I wonder if there was not an irregularity in the laying down of the mucosa in the lower part and if it was not really esophagus instead of stomach.

DR. CHURCHILL: Of course the short esophagus theory has been advanced as a reason for not operating on hernia of the esophageal orifice. In the ones we have seen at operation the stomach comes down out of the hernial pouch and the cardiac orifice is in the normal place. We have not seen any instance of the so-called short esophaguses at operation.

DR. RICHARDSON: I may say that clinically when I first saw the patient I thought he had carcinoma somewhere and was reluctant to accept a diagnosis of duodenal ulcer. After the x-ray diagnosis however the symptoms became much more typical of it.

His history is divided into three periods, the first one when he did well on ulcer treatment; the second one, eight months ago, when he did poorly on every type of ulcer treatment I could think of, and where I again thought there was some process other than ulcer to account for the symptoms. There began then to be a prominence of the mid-dorsal pain which he complained of in the beginning. Then finally and suddenly, at least to me suddenly, came the symptoms of esophageal obstruction, which did not appear until about two weeks before I saw him and perhaps three weeks before his entry to the hospital. Then I thought in spite of the x-ray findings that he had carcinoma of the esophagus.

DR. D. CAMPBELL SMYTH: May I say a word? I esophagoscoped this patient. The findings were typical of a healed ulcer of the esophagus about two inches above the cardiac, and I should be very much surprised if there was any malignancy in this region. The esophagus was very much closed down and it was impossible even to get a bronchial bougie through the constriction. There was slight bleeding of the posterior wall which gave me the idea that there might be slight activity of the ulcer. The late Dr. Morrison had a large series of cases of hernia of the stomach along the side of the esophagus and he promised from year to year to publish them, but never did.

I should like to ask Dr. Hampton whether the patient was sitting down or standing.

DR. HAMPTON: He was standing, and no barium passed through the esophagus for the first five or ten minutes. Then we added as much water as he could drink. You can see the level of the barium where it is in contact with the water.

CLINICAL DIAGNOSES

Ulcers of the esophagus (obstructing) and of the duodenum.
Diaphragmatic hernia.
Bronchopneumonia.

ANATOMIC DIAGNOSES

Stricture of the esophagus, probably healed ulcer.
Diaphragmatic hernia.
Penetrating ulcer of the duodenum.
Chronic gastritis.
Acute mediastinitis.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The postmortem examination showed a marked degree of stricture of the esophagus, the lumen being narrowed to two millimeters in diameter at the most marked point. Above that area the esophagus was slightly dilated. There was nothing grossly to suggest malignancy, and microscopic examination completely rules it out. The entire esophageal wall shows a marked chronic inflammatory process with a great deal of scarring of the submucosa. The epithelium has entirely disappeared, so that I do not think that histologically it is possible to say whether or not this ulcer occurred in an island of gastric epithelium.

In addition there was a mediastinitis as a terminal infection, though no area of perforation in the esophagus could be demonstrated.

The duodenum showed a typical deep penetrating ulcer on its posterior wall at the point where it had been seen by x-ray. It had burrowed well into the head of the pancreas, miss-

ing the pancreaticoduodenal artery by only one millimeter.

The lungs showed a moderate degree of collapse and a very small trace of pneumonia. The other organs were all essentially negative.

DR. HOLMES: How far above the cardiac orifice was this?

DR. MALLORY: It was about 3 centimeters.

DR. BOCK: Did the gastric mucosa in the herniated portion of the stomach show any change?

DR. MALLORY: The entire stomach showed very severe gastritis from top to bottom, no essential difference above or below the hernia.

DR. BOCK: How much of the stomach was above the diaphragm?

DR. MALLORY: About three centimeters of it.

DR. BOCK: Not much of a pocket then?

DR. MALLORY: No, not a very large one. We did not measure the esophagus, but we got the impression that it was short. In the other cases of diaphragmatic hernia that I have seen the esophagus has looped around and has come into the cardia. That condition was not present here. With such an extreme degree of scarring it is reasonable that the esophagus might have been contracted as well as narrowed by the inflammatory process.

DR. BOCK: Was the gastritis the sort of thing we associate with ulcer of the duodenum?

DR. BOCK: Yes, although a little more diffuse than usual.

Have you anything to add, Dr. Mosher?

DR. MOSHER: No, except that I came near saying this man would die of mediastinitis. I have said before that every examination of the esophagus, however simple, is a potential tragedy, and when you go down even to remove a specimen, the smaller the growth the greater is the danger in removing that specimen. If there is a fungating growth and you can take that off you can get by; but if you have a very low growth and try to get that off you run the danger of perforation. These are the cases which put the Throat Department in bad with the Medical side.

DR. CHESTER M. JONES: I should like to ask Dr. Mosher a question if I may. In a case where one pretty strongly suspects esophageal ulcer with as much stricture as in this case, is it ever conceivable that the passage of bougies might cause damage by stretching an ulcerated area?

DR. MOSHER: I should be afraid of it. I should attempt with what residual skill I have left to dilate cautiously, but it is a very dangerous procedure because of the danger of splitting the scar and starting up a mediastinitis.

CASE 20062

PRESENTATION OF CASE

DR. J. MARK HIEBERT:* A white male infant four days old was admitted to the hospital by transfer from a suburban hospital where he was born. The delivery was normal. The birth weight was 5¾ lbs. Nothing abnormal had been noted until fluids were offered: then almost immediately they were spit up. Barium feeding was given and x-rays showed an atresia of the esophagus. Fluids and whole blood were given subcutaneously. The child was sent to the Massachusetts General Hospital for possible operation.

*Interne on the Children's Medical Service.

The child appeared normal for his age and in a fairly good condition. The temperature was 98°, the pulse 130. The respirations were 55. In the process of examination he suddenly became cyanotic. Mucus was quickly cleared from the throat and oxygen and artificial respiration were given. The throat was filled with a rather sticky mucus. The nose was obstructed with a similar discharge. The lungs were normal to percussion, but many coarse rattles and rhonchi were heard on auscultation. The heart appeared normal.

Other attacks of cyanosis occurred during the next sixteen hours; these were relieved by oxygen inhalations. X-ray examinations of the chest were repeated in this hospital. On the day following admission an operation to relieve the abnormalities was done. Following the operation there was some bleeding from the mouth. The child died twelve hours after operation.

CLINICAL DISCUSSION

DR. HAROLD L. HIGGINS: Our clinical diagnosis was a congenital atresia of the esophagus.

In these cases of congenital atresia of the esophagus we also find in practically every case a connection of the lower end of the esophagus with the trachea. There is a sac at the upper end of the esophagus and a connection between the lower end of the esophagus and the trachea.

In palpating the abdomen in these children one is likely to cause coughing. This child did not show such striking coughing as I have seen in some other children.

We felt that the lung signs in this case were probably due to aspiration of material that came from the stomach by the esophageal-tracheal fistula. We felt very certain that there was a connection between the trachea and the esophagus from the fact that air was seen in the stomach by x-ray. We did not think that there was such a large opening between the esophagus and the trachea as usual because the child was considerably more free from attacks of cyanosis and coughing.

The prognosis in cases of this nature has been almost universally bad. I know of no cases that have lived more than two weeks. Dr. Morse in his book recommends that these children be allowed to die in peace. However, we could not quite decide to follow Dr. Morse's recommendation and the child was referred to Dr. Linton

and Dr. Churchill to see if something could not be done to give him a useful esophagus.

DR. GEORGE W. HOLMES: We saw this child in the X-Ray Department and he created a great deal of interest because we found definite obstruction in the esophagus and air in the stomach. We did not understand how the air could reach the stomach if the esophagus was absent in its lower part. After consulting with Dr. Higgins we found how this happened.

The film also shows a small amount of barium in the bronchial tree, and we were able to demonstrate a stricture of the esophagus, a broncho-esophageal fistula and air in the stomach.

DR. TRACY B. MALLORY: Dr. Bremer has been kind enough to come down and I hope he will tell us something about the embryological development of these conditions.

DR. J. L. BREMER: The difficulty in understanding these cases lies in the lack of knowledge as to how the lungs develop. We think of them as glands, and we expect a gland to grow out from a duct, the duct to branch, and the end-pieces to develop at the ends of the branches. That is true of most glands, though certain of them lose their ducts or change in one way or another. The lungs do not grow in that way. The lung is the gland itself, the main bronchus is its duct, and the trachea, which connects the two lungs with the pharynx, is part of the original pharynx, not an outgrowth from it.

If we draw a side view of the pharynx in an early embryo, beginning at the mouth and extending downward (Figure I), we find the

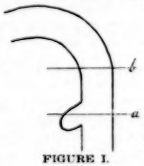

FIGURE I.

lungs developing as branches or side-pocketings in the position of *a*, and that is away up in the neck. The rest of the tube continues down to the stomach. Then there is a change of relative position, with a great lengthening of the pharynx between *a* and *b*, as in Figure II. The lung buds are carried relatively much nearer the stomach. The trachea is formed not by an outgrowth in the position of the lung buds, but by a subdivision of the single pharynx above this point into two tubes, trachea in front and esophagus behind, by the fusion of two longitudinal ridges, pressing the lateral walls of the pharynx. The ridges develop normally along the dotted lines of Figure II, causing the complete separation of trachea and esophagus. Partial failure of the fusion may lead to anomalies, as may also the unusual position of the ridges. Failure to close in the region of *a* would give a connection between the trachea at its lower end and the esophagus. In that case the child could get both air and food, but there would always be the possibility of food being regurgitated into the lungs or of air getting into the stomach. If the position of the ridges is abnormal so that they cross the lower part of the future esophagus (line of dashes, Figure II) we have exactly the condition in the case under consideration,

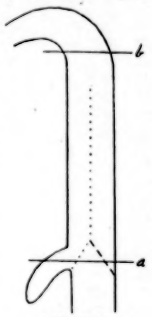

FIGURE II.

the trachea opening into the bronchi and also into the lower esophagus, while the upper esophagus ends blindly.

The condition arises very early. Trachea and esophagus should be fully separated in embryos of ten millimeters and Dr. F. T. Lewis has discovered one of about that age which already showed an anomaly like that in the present case. I believe some operations for the remedy of the condition have been tried, approaching from the back, tying the trachea-esophagus connection, and attempting to reconstruct the esophagus, but I do not know how successful they have been.

DR. MALLORY: Dr. Churchill made an attempt to reconstruct things here. I hope he will tell us what he tried to do.

DR. EDWARD D. CHURCHILL: One thing in regard to the diagnosis: It is probably not advisable to give these babies barium. If there is any doubt in your mind about the diagnosis it is better to use a little lipiodol rather than barium, because the barium goes directly into the lungs and adds to the dangers of pneumonia. This child did have pneumonia at the time we operated. The diagnosis can usually be made when a baby is unable to swallow and the stomach is found distended with air. The barium comes from the blind end.

What we found at operation was just what we had pictured. The upper end of the esophagus ended blindly about the level of the first or second thoracic vertebra above the arch of the aorta. The lower end of the esophagus departed from the trachea at a point just above its bi-

furcation and there was a rather attenuated cord leading down to a normal esophagus and then the stomach. Of course the air in the stomach had come through this route. The barium in the lungs had come from an overflow of the blind upper end into the pharynx and down the trachea. It is a difficult problem for surgery to handle. If you do a gastrostomy any food placed in the stomach will run up into the lungs.

In operations at the Children's Hospital, where they have had an extensive experience, they have attempted to bring both ends of the esophagus out through the back. Dr. Lanman has had a case of a child who lived nearly forty-five days after bringing the esophagus out and feeding through a tube. It seems to me that the difficulties of bringing up a child with the two ends of the esophagus in the back and later getting any kind of reconstruction of the two in the period of growth of the child are a little too much to contemplate. In my inexperience I decided to attempt here the all-or-none procedure of getting restoration of the esophagus in the mediastinum. We were able to go through the pleural cavity into the mediastinum, disconnect this from the trachea, close the fistula, and then using the attenuated fistulous tract to fix the upper end and pull it down a bit. An opening was made in the upper segment and a small Levine tube inserted up until it came out of the mouth. The other end of the tube was inserted into the lower end of the esophagus and entered the stomach. The mediastinal tissues were closed over the tube for the space of about one inch that lay free in the mediastinum. It looked all right when we got through. Of course a rubber tube running down the middle of the mediastinum is not very physiologic.

The child stood the operation beautifully with intratracheal anesthesia given by Dr. Bradshaw. The child left the table in excellent condition and died that night, as we expected.

DR. HARRIS P. MOSHER: Would you do it again, Dr. Churchill?

DR. CHURCHILL: I think I should. The only alternative for the infant is certain death from starvation.

CLINICAL DIAGNOSES

Atresia of esophagus, congenital.
Cachexia.
Bronchopneumonia?

ANATOMIC DIAGNOSES

Congenital atresia of the esophagus.
Broncho-esophageal fistula.
Bronchopneumonia.
Operation wound, attempted repair of atresia of the esophagus.

PATHOLOGIC DISCUSSION

DR. MALLORY: The autopsy showed that everything had been carried out as Dr. Churchill described. The mediastinal tissues had been pulled around the rubber tube so as completely to encircle it, and it looked possible that at some later period if the child had survived the acute stage the rubber tube could have been removed and a tube-like passage would have been left. The story would not be over then, but at least he would have had a passageway from his mouth to his stomach.

The immediate cause of death was not entirely obvious. The lungs had been quite successfully reëxpanded and there was no significant degree of collapse. There was considerable edema of the pulmonary tissues and there was a slight degree of bronchopneumonia. I think that that was the immediate cause of death, on top of the general shock of the operation to so young a child.

DR. HOLMES: Dr. Churchill has raised two questions about which I would like to speak. This is the shadow that I pointed out and made the remark that the barium had passed from the esophagus into the bronchi. It is also possible that the child regurgitated the barium and it passed into the trachea in this way. The finding of barium in the bronchi does not prove that it entered through an artificial opening.

The other point that he raised was the danger of giving barium in such a case. I would disagree with him on that point. I do not think it is any more dangerous than food, which had already been given. I remember vividly the first case that I saw of an adult with an esophageal fistula. The patient drank barium and I saw the whole bronchial tree fill. After I had advised that he be put on the danger list it occurred to me that his food had been doing the same thing for some time and that he would cough it up without trouble. In the absence of the cough reflex that might be dangerous, but as a rule one does not have to worry about barium going into the bronchial tree.

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O TEMPORA! O MORES!

IN comparing the status of the medical profession today with that of a century ago, we are accustomed to believe that our medical forefathers occupied a more enviable position. We visualize a dignified old gentleman with gold-headed cane, whose word was law and whose relation to his patients was that of a benevolent despot.

A very different picture is called forth by a pamphlet entitled "Medical Morals", published by one George Gregory in 1853. Mr. Gregory was impelled to write this treatise because "the evil which it aims to expose and correct is so inveterate, so vast and appalling, as to justify any needful remedy." This evil, in brief, was the examination of women for pregnancy and "female disorders" by male members of the profession. "The establishment and preservation of chaste and delicate customs in society, is an object in which every good man and woman must necessarily feel a deep interest." Submission to physical examination ruins the beautiful flower of modesty and leads to seduction and adultery. "God has decreed that every man shall have his own wife free from mercenary or other pollution, and no tampering of the medical faculty can for a moment be permitted without destruction to the marriage compact."

To prove the heinous nature of this "violation of the instincts of nature", Gregory quotes passages from current medical authors, which describe the art of vaginal examination by "touching" and by sight. The use of a lighted candle to aid the examiner in his inspection of the cervix is particularly condemned, as being the greatest possible outrage to woman's modesty. Two woodcuts, taken from "Magrier's Midwifery Illustrated", serve to drive home to the lay reader the enormity of this outrage. One cut depicts an acidulous female in standing position; her physician, one knee upon the floor and a lascivious expression on his face, has both hands beneath the patient's skirts.

The remedy for this deplorable state of affairs, Mr. Gregory believed, consisted in the establishment of female medical colleges for the training of midwives and women doctors. Such a college—the New England Female Medical College—had been opened in Boston in 1848, but lacking in funds, it had withered at the root. In 1851, the Committee on Education of the Massachusetts Legislature reported in favor of a grant of money to this institution, saying, "It is, moreover, a matter of grave importance to encourage such customs in the community as shall not tend to weaken, but rather cherish a proper reserve between the sexes. . . ."

In the light of twentieth century ideas, one cannot read the fervent passages of Mr. Gregory without a smile, yet evidently a good many people felt as he did one hundred years ago. The tremendous swing in the attitude of the public toward an acceptance of scientific ideals is an astounding phenomenon. We, members of the maligned profession, may feel that this change is gratifying testimony to our "medical morals", and to our ability to maintain an impersonal, scientific attitude toward our work, in spite of those temptations and opportunities which Gregory paints with so lurid a brush.

ARE PHYSICIANS RESPONSIBLE FOR THE HIGH MATERNAL MORTALITY?

"MEDICAL Council Approves Report Blaming Doctors for 61 per cent of Mortality" is a headline appearing in a well-known daily newspaper which is neither red nor yellow. It is maternal mortality at childbirth that is meant and the natural conclusion from the pronouncement is that if these patients had not placed themselves in the care of the physicians they would not have died. Is there any surprise that objection has been made to the publicity which has been given to the report of the sub-committee of the New

York Academy of Medicine which has made a study of maternal mortality to determine if possible if there were evils for which medical and obstetrical practitioners might be responsible? Yet it is not the publicity but the inaccuracy that is objectionable, for just below in the text is the statement that 66 per cent of the mothers who died in childbirth could have been saved by the application of medical knowledge, and that more than 61 per cent of these avoidable deaths were directly attributable to the medical profession. Thus on its own showing only 40 per cent instead of 61 per cent of the total are attributed to the profession. It is not likely that even this conclusion will receive widespread acceptance, without a good deal of qualification, by those who know, but it and other unjustifiable conclusions are likely to be drawn by the ignorant.

There are two criteria in the light of which results may be judged. They are not so widely separated as appears at first thought. The philosopher says "Where attainable knowledge could have changed the issue, ignorance has the guilt of vice." The jurist says "the physician shall use reasonable care" which takes into consideration time, and place and circumstance.

Several questions must be asked, and they should be answered only after due knowledge is available. What is the evidence that, in general, physicians are not practicing in accord with the best knowledge and practice of today? What is the evidence that physicians are not living up to the teachings they received in medical school? Are they not doing the best they know how? What are the discrepancies between what the physician was taught in school and the best thought and practice now? Why have physicians who practice obstetrics not kept up with the times? Are practicing physicians in other fields abreast with the times, and if not why has not attention been directed to them also? How much blame should be placed on the physician for not giving the patient the best possible care, if he does the best he knows how?

The report by the New York Academy of Medicine is by no means a satisfactory answer to these questions and perhaps amounts to little more than this: there is a considerable number of women of whom the medical treatment provided cannot be characterized as the best that medical knowledge and skill can furnish, and because of this lack they die. This is true of other fields than obstetrics, but the complication of infection in childbirth is especially likely to have a fatal outcome.

It is a fact, that considering the state of medical knowledge about the processes of childbirth and what can be accomplished by the most skillful and wise obstetrician, the equipment of the ordinary physician is inadequate. Here is a serious deficiency in our medical education. The surgical risks of childbirth are not properly appreciated by physician or layman. The growing emphasis on the value of the life of the child has tended to produce underestimation of the risk to the mother. There is a tendency, too, to insist that every patient have the best medical care, irrespective of ability to pay for it, and just at present there persists a strong tendency to report gross figures in maternal mortality without discriminating and constructive criticism, that may lead directly to real improvement in the situation.

In one sense far more is known about the prevention of tuberculosis than of puerperal infection. Why is tuberculosis not wiped out after this half century of struggle? Theoretically, whatever that may mean, the problem is simple, but deaths from tuberculosis are still numerous though the rate is diminishing. Not even theoretically is the maternal deathrate problem simple. What is needed is painstaking, thorough, intensive, scientific study of the problem from all sides, with heroic restraint, so as not to draw conclusions before adequate evidence is available.

The late J. Whitridge Williams said years ago, after one of his patients had died of sepsis: "I am responsible for her death, but I do not know how or why and I would treat another patient in exactly the same way." Later he was more convinced of the existence of autogenous sepsis following childbirth, but the example of his fine courage is a beautiful tradition.

In one sense it is true that the medical profession is responsible for the high maternal mortality, but this statement needs so many qualifications that if it is put forth baldly it becomes untrue. Certainly it is not true that because a woman dies in childbirth, her physician is to be blamed.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MUNRO, DONALD. A.B., M.D. Harvard University Medical School 1916. Visiting Surgeon in charge of Neurosurgery, Boston City Hospital. Assistant Professor of Neurological Surgery, Harvard Medical School. His subject is "The Diagnosis, Treatment and Immediate Prognosis of Cerebral Trauma. An Introductory Study of 1494 Cases." Page 287. Address: Boston City Hospital, 818 Harrison Avenue, Boston.

FOSTER, JOHN H. B.S., M.D. University of Pennsylvania School of Medicine 1917. Associate Medical Professor, Hunan-Yale Medical School, Changsha, China 1919-1927. Attending Physician, Waterbury (Conn.) Hospital. His

subject is "Amebiasis in Connecticut." Page 294. Address: 103 North Main Street, Waterbury, Connecticut.

CHAMBERLIN, H. A. M.D. Tufts College Medical School 1913. F.A.C.S. Professor in Urology, Tufts College Medical School. Urologist, Boston Floating Hospital. Visiting Urologist, St. Elizabeth's Hospital, Brighton. Surgeon-in-Chief, Genito-Urinary Department, Boston Dispensary. Address: 520 Commonwealth Avenue, Boston. Associated with him is

MACMAHON, H. E. A.B., L.M.C.C., M.D. University of Western Ontario 1925. Professor of Pathology and Bacteriology at Tufts College Medical School. Address: 31 Pinckney Street, Boston. Their subject is "Papillary Carcinoma of the Renal Pelvis." Page 299.

SWEET, RICHARD H. M.D. Harvard University Medical School 1926. F.A.C.S. Assistant in Surgery, Massachusetts General Hospital. Visiting Surgeon, Palmer Memorial Hospital. His subject is "Uterus Didelphys." Page 303. Address: 205 Beacon Street, Boston.

BENNETT, DARWIN E. B.S., M.D. Harvard University Medical School 1931. Surgical House Officer, Peter Bent Brigham Hospital January 1, 1932-May 1, 1933. His subject is "Recurrent Trichobezoar." Page 307. Address: Methodist Episcopal Hospital, Brooklyn, New York.

MARLOW, F. W. M.R.C.S., L.S.A., M.D. Syracuse University College of Medicine 1885. F.A.C.S. Emeritus Professor of Ophthalmology, Syracuse University College of Medicine. Ophthalmologist to the Syracuse Memorial Hospital. His subject is "The Symptoms of Hidden Ocular Muscle Imbalance." Page 309. Address: 1003-4 State Town Building, Syracuse, New York.

MYERSON, ABRAHAM. M.D. Tufts College Medical School 1908. Professor of Neurology, Tufts College Medical School. Director of Research, Boston State Hospital. Visiting Neurologist, Boston City and Beth Israel Hospitals. Address: 270 Commonwealth Avenue, Boston. Associated with him is

LOMAN, JULIUS. M.D. Tufts College Medical School 1925. Instructor in Neurology, Tufts College Medical School. Psychiatrist, Research Staff of Boston State Hospital. Junior Visiting Neurologist, Beth Israel Hospital. Address: 270 Commonwealth Avenue, Boston. Their subject is "Progress in Neurology, 1932." Page 314.

MASSACHUSETTS LEGISLATIVE NOTES

H. 509 is an act regulating the administering of ether, chloroform, nitrous oxide gas or other substance or gas producing unconsciousness, and reads as follows: The practice of medicine shall include the administering to human beings of ether, chloroform, nitrous oxide gas or other substance or gas producing unconsciousness, except when administered by a registered dentist.

H. 1156 is an act requiring physical examinations of persons engaged in the handling of food for public consumption, and reads as follows: All cooks or other persons in any way connected with the preparation of food for consumption in a hotel, inn, restaurant or other public place shall undergo semi-annually physical examinations by duly registered physicians to determine whether they have any communicable disease.

H. 1155 is a resolution providing for an investigation and report on the effect of gas from motor vehicles on the health of the public, and reads as follows: *Resolved*, That the department of public health is hereby directed to investigate the effect of the exhaust gases from motor vehicles on the public health and to make its report to the general court with such recommendations for legislation as it may deem proper, accompanied by drafts of such measures as it may recommend, on or before March fifteenth of the current year.

H. 919 is an act to provide for a clinic to be administered by the State Department of Public Health for treating alcohol addicts.

MISCELLANY

AMERICAN MEDICAL ASSOCIATION

To Secretaries of State Medical Associations:

The following instructions have been issued by the United States Compensation Commission, relative to the hospitalization of employees of the Federal Civil Works Administration.

(Signed) WM. C. WOODWARD,
January 10, 1934 *Legislative Counsel.*

UNITED STATES EMPLOYEES' COMPENSATION COMMISSION

Washington

January 8, 1934.

From: United States Employees' Compensation Commission

To: State Civil Works Administrators

Re: Selection of hospitals in compensation cases arising out of injuries to employees of the Civil Works Administration.

Please instruct each local administrator in your state as follows:

1. Employees of the Civil Works Administration who suffer injuries while in the performance of duty are entitled to necessary hospital care for the treatment of conditions due to such injuries. An injured employee shall be admitted to and retained in a hospital only as long as hospitalization is necessary for the purposes of treatment or examination. The instructions herein prescribe the procedure to be

followed in selecting hospitals for the treatment of these cases and the schedule attached shows the rates to be allowed for hospital care. In no event, however, should these instructions be construed so as to interfere with the prompt and adequate care of an injured employee.

2. Injured employees must be referred to Federal hospitals when such hospitals are both available and adequate. It is not intended to utilize these governmental facilities for civil works employees to the disadvantage of other classes of beneficiaries that may be entitled to care in Federal hospitals, but that beneficiaries for whom the respective Federal hospitals were primarily established shall have preference in the use of such hospitals. However maximum use should be made of any existing Federal medical facilities that may be available.

3. (a) When Federal hospital facilities are not available or adequate, cases requiring immediate hospital care shall be sent to the nearest suitable hospital which desires to participate in the service at the rates specified in the approved schedule of rates. Public hospitals, other than Federal, are not to be given preference.

(b) The following factors should be considered in determining suitability: the proximity of the hospital, type of service, e.g., whether the hospital is well qualified to handle the special type of case, and the general quality of service.

(c) You should secure advice as to the suitability of local hospitals from one or more of the following local sources: medical advisory councils which may already be set up under Rules and Regulations No. 7, of the Federal Emergency Relief Administration; hospital associations; hospital, health or similar councils; county medical societies; boards of public welfare or health.

4. (a) All hospital care must be authorized in writing by the proper officials on the staff of the local Civil Works Administrator. Care of emergency cases should not be delayed for a written authorization, but this must be furnished within 48 hours after admittance to a hospital.

(b) An authorized physician in charge of the treatment of an injured employee as a compensation patient when hospital care is required may send the patient to a hospital of the physician's selection provided the hospital thus selected agrees to the approved schedule of rates. However, hospitalization in such cases must be approved in writing as provided in paragraph 4 (a).

5. The Commission reserves the right to have its medical representatives examine patients at the hospital and examine the records of these patients and to cause the patient's removal when the Commission considers it necessary in the interest of the patient or to prevent overcharge, or for other sufficient reason. Hospital records of these patients shall be open to inspection by representatives of the Commission.

6. The Commission in conference with representatives of the National Hospital Associations has agreed on a basic rate for the care of injured Civil Works Administration employees in general hospitals, exclusive of Federal hospitals. This rate includes many items for which extra charges are usually made. A schedule of rates for other services which are not included in the basic rate has also been agreed to. The National Hospital Associations have agreed to notify their members of these approved rates and urge their full coöperation with the Commission. The approved schedule of rates is attached hereto. Charges for services previously rendered will be adjusted under this schedule. (See paragraphs 34 and 35 of Civil Works Administration Rules and Regulations No. 5 for instructions concerning submission of vouchers.)

7. Each local administrator must make adequate provisions for the transportation of seriously injured employees to obtain medical treatment, by arrangements made in advance for each work project. This may be done by arrangements for the use of automobiles available at the project, by agreement concerning the use of local ambulance service or such other arrangements as may be feasible. Ambulance services provided by hospitals is covered in the approved schedule of rates.

U. S. Employees' Compensation Commission.

Schedule of Hospital Fees Agreed Upon Between the Joint Committee of the American, Catholic and Protestant Hospital Associations, the Civil Works Administration and the U. S. Employees' Compensation Commission

A $3.50 per diem rate for all hospital cases of injured employees of the Civil Works Administration will be general throughout the United States, regardless of local hospital costs or charges. This rate will apply in general hospitals, exclusive of Federal.

The following items will be included in the rate:

The use of a single room when necessary
General medical and surgical care by the house staff

Ordinary nursing	Material for plaster casts
Special diets	Colonic irrigations
Usual medicines	Hypodermoclysis
Usual dressing and surgical supplies	
Usual laboratory tests, such as:	
Blood counts	Coagulation time
Smears	Haemoglobin estimation
Usual urine tests	Occult blood
Wassermann tests	Skin tuberculin tests
Precipitation tests for syphilis	Spinal fluid smears and cell counts
Widal tests	Sputum examination for tubercle bacillus
Agglutination tests	
Blood typing	
	Other usual bacteriological tests

Such physiotherapy treatments as may be necessary for patients in the hospital

Autopsies and reports of same when a patient dies in the hospital

There will be no charge for medical or hospital reports unless an actual transcript of the hospital record be requested, in which case charge for same will be made in accordance with the local public stenographers' rates.

Charges will be allowed for the day of admission, but not for the day of discharge or death.

In addition to the above rate it will be permissible to make the following extra charges:

(1) An operating room fee of $5.00 for a minor operation and $10.00 for a major operation. A general anesthesia fee of $5.00 for a minor operation and $10.00 for a major operation, to include anesthetic service by a salaried employee of the hospital and the cost of the anesthetic.

(2) Laboratory examinations of an unusual character, such as complete blood chemistry; gastric analyses, etc., may be charged for at a rate of from $3.00 to $5.00, according to the nature of the examination (which must be specified in the voucher submitted), $3.00 being the usual charge allowed for such examinations and reports.

(3) Fee for special nursing when necessary will be allowed in accordance with the local prevailing rate or when furnished by a salaried employee of the hospital, at actual cost.

(4) X-ray examination will be paid for in accordance with the following rate, the number of films and procedure for each fee being indicated by the description below:

	No. of films	Price
Ankle joint, antero-posterior and lateral views	2	2.50
Arm, humerus, antero-posterior and lateral views	2	2.50
Bladder, with injection, antero-posterior views	1	5.00
Chest, for pulmonary or cardiac diagnosis, plain	1	3.75
Chest, for pulmonary or cardiac diagnosis, stereoscopic	2	5.00
Clavicle, postero-anterior view	1	2.50
Elbow, antero-posterior and lateral views	2	2.50
Fluoroscopy, when required, without film	1	1.00
Foot, antero-posterior and lateral views	2	2.50
Forearm, radius and ulna, antero-posterior and lateral	2	2.50
Foreign body in eye, location of (the fragment charted in three planes and its dimensions ascertained by the method of Sweet or equivalent as needed)		12.50
Gall bladder, Graham technic, including cost of dye	1	10.00
Gastro-intestinal tract, complete x-ray study, including fluoroscopy, as needed		12.50
Hand, antero-posterior and lateral views	2	2.50
Hip joint, plain, antero-posterior view	1	3.75
Hip joint, stereoscopic, antero-posterior view	2	5.00
Intestine, barium clysma, 14 x 17 films for position and outline, as needed		7.50
Jaw, upper or lower	1	2.50
Kidneys, right and left, for comparison, 11 x 14 films, as needed		5.00
Knee joint, antero-posterior and lateral views	2	2.50
Leg, tibia and fibula, stereo-posterior and lateral views	2	2.50
Lipiodol injection for bronchiectasis, etc., including roentgenograms and interpretation, as needed		12.50
Pelvis, 14 x 17, single film, antero-posterior view	1	5.00
Pyelography, using uroselectan or similar preparation (including cost of drug)	4	10.00
Ribs, plain view over suspected area, 10 x 12 film	1	3.75
Scapula	1	2.50
Shoulder joint, plain, antero-posterior views	1	2.50
Shoulder joint, stereoscopic, antero-posterior views	2	5.00
Sinuses, frontal and ethmoid, antero-posterior and lateral views	2	5.00
Sinuses, mastoid, right and left sides for comparison	2	5.00
Sinuses, maxillary, antero-posterior and lateral views	2	5.00
Skull, ventriculogram — air injection — as needed		7.50
Skull, antero-posterior and lateral views	2	5.00
Skull, stereoscopic	2	7.50
Spine, cervical, antero-posterior and lateral views	2	5.00
Spine, dorsal, antero-posterior and lateral views	2	5.00
Spine, lumbo-sacral, with coccyx, antero-posterior and lateral views	2	5.00
Stomach, barium or bismuth meal, 14 x 17 film, after ingestion four 8 x 10 films for detection of duodenal cap; total of four 8 x 10 films, including fluoroscopy	4	12.50
Teeth, single film	1	1.00
Teeth, each additional film up to and including five films	1-5	1.00

Teeth, series, (five films up to and including full mouth)	over 5	5.00
Thigh, femur, antero-posterior and lateral views	2	3.75
Ureters, right and left, for comparison	1 or 2	7.50
Wrist, antero-posterior and lateral views	2	2.50

(5) Unusual expensive medication and appliances will be supplied at cost. This includes such items as oxygen administration (marked preference being given to the use of commercial oxygen) biologicals; prosthetic and orthopedic appliances, when furnished by the hospital. Blood transfusions not to exceed $5.00 per 100 cc. to donor, and a hospital charge of $5.00 for the transfusion as a minor operation will be allowed.

(6) Ambulance charges when furnished by the hospital may not exceed a minimum rate of $3.00 when the call is within a three mile radius of the hospital. An additional rate of 50c per mile beyond the three mile radius, one way, will be allowed.

(7) Professional and other fees of persons not employed by the hospital are not included in this agreement.

(8) Fees for hospitalization, and prophylactic treatment of contagious diseases not ordinarily treated in general hospitals are not included in this agreement and should be subject to local regulation.

CORRESPONDENCE

THE RESTORATION OF THE REGISTRATION OF DR. DWIGHT F. WILLIS

Editor, New England Journal of Medicine,

This is to inform you that at a meeting of the Board of Registration in Medicine held January 25, 1934, it was voted to restore to Dr. Dwight F. Willis his license, suspended September 28, 1933.

Yours very truly,

STEPHEN RUSHMORE, M.D., *Secretary.*

RECENT DEATHS

HOWE—JOSEPH DIMOCK HOWE, M.D., of Pittsfield, Massachusetts, died in that city January 30, 1934, after a two weeks' illness. Dr. Howe was born in 1869 in New Baltimore, New York, and graduated in medicine from the Tufts College Medical School in 1898.

He joined the Massachusetts Medical Society in 1899, and was a Fellow of the American Medical Association. He had served as assistant medical examiner of his district for fifteen years.

During the Spanish-American War he was a Y. M. C. A. worker at Chicamauga, and held membership in Richard H. Dowling Camp, Veterans of the Spanish-American War. He served as a member of the Pittsfield School Board in 1915-1916. Previous to settling in Pittsfield, he practiced in Becket and Cheshire. He was a Mason and was affiliated with the Congregational Church.

Surviving relatives include a sister, Winifred Hayden Howe, of Pittsfield; a brother, Ralph Barnard Howe, of Schenectady, New York, and several nieces.

RIOPELLE — ALEXANDER JOSEPH RIOPELLE, M.D., who had practiced in Lawrence, Mass., for forty-five years, died at the home of his daughter, Mrs. Adelard G. Valcourt, 75 Tremont Street, Lawrence, January 26, 1934. He is survived by five daughters, three sons and a sister.

BOOM — AUGUSTUS KEEFER BOOM, M.D., of 51 Park Street, Adams, Massachusetts, died January 24, 1934. Dr. Boom was born at Albany, New York, May 13, 1866, son of James and Lucy Boom. He was graduated from the Cleveland College of Physicians and Surgeons in 1888 and settled in Adams that same year. He joined the Massachusetts Medical Society in 1897. He served as president of the Berkshire District Medical Society in 1916. He served as selectman of Adams for three terms and for a part of these periods was chairman of the board. He was later a member of the Board of Health and town physician. He was a member of the staff of the North Adams and the Plunkett Memorial hospitals. He was an examiner for the Metropolitan Life Insurance Company for many years. He is survived by his widow, Mrs. Myria S. (de Rouville) Boom; two daughters, Miss Hazel and Miss Florence Boom; also his mother, living in Albany, New York; a sister, Mrs. Joseph Jewell, and a brother, James Boom.

OBITUARIES

FRANK LINDEN RICHARDSON, M.D.

In the death of Frank Linden Richardson, the Boston Society of Anesthetists has lost one of its original and most valued members, a former President, and the Dean of Anesthesia in Boston.

Born in 1877 he died suddenly of coronary occlusion on November 16, 1933. He was a special student at the Massachusetts Institute of Technology; he received his M.D. degree from the Harvard Medical School; and he interned at the Boston City Hospital. His work in anesthesia won him wide recognition from the surgeons of Greater Boston, perhaps especially from those doing nose and throat work. This recognition of his work is evidenced by the positions which he held, such as chief of the Anesthesia Service at the Boston City Hospital, Consultant in Anesthesia at the Children's Hospital, and Lecturer in Anesthesia at the Tufts College Medical School.

He was also the first president of the Eastern Society of Anesthetists.

In his professional work he held a nice balance between progressiveness and conservatism. His judgments were sound and highly valued. Perhaps the most outstanding characteristic of his work was its nicety of detail. There was never any suggestion of crudeness or roughness. He loved fine mechanics, and had both at his office and at his home a jeweler's lathe and other fine tools, with which he loved to work. His ether vaporizer, almost universally adopted by the anesthetists of Boston, was a product of this work.

Many of the members of this Society have reason to remember the generous welcome he gave them when they entered the specialty, and a number owe him eternal gratitude for his very real help in their early struggles in anesthesia. The Society indeed mourns the loss of a very real friend.

L. F. S.

RESOLUTIONS OF THE SENIOR STAFF OF THE BOSTON CITY HOSPITAL IN APPRECIATION OF DR. FRANK L. RICHARDSON, LATE VISITING ANESTHETIST.

Dr. Frank Linden Richardson of Newtonville, Massachusetts, died suddenly on November 16, 1933. He was born in Concord, Massachusetts in 1877. He graduated from the Massachusetts Institute of Technology and then entered Harvard Medical School, graduating in 1903. He is survived by his widow, Mrs. Constance Mathey Richardson, and two sons, Paul H. Richardson and David A. Richardson, both of whom are students at the Massachusetts Institute of Technology.

He served an interneship in the surgical department of the Boston City Hospital from 1904 to 1906 and became head of the Anesthesia Service in 1910. He was a consultant in anesthesia at the Children's Hospital and Beth Israel Hospital and lecturer on anesthesia at Tufts Medical School.

Among his earlier publications was one written in conjunction with the late Dr. Edward H. Nichols on Arthritis; this paper was an outstanding contribution and is still regarded as the model description of this disease. His later publications included: Some Modern Ideas About Anesthesia, Heart Lesions in Anesthesia, Anesthesia for Minor Surgery, Anesthesia for Prostatectomy, Some Suggestions for the Dietetic, Pre-Operative and After-Care of Surgical Cases. His societies included: The Massachusetts Medical, American Medical Association, Boston Society of Anesthetists, International Anesthesia Research Society, Associated Anesthetists of the United States and Canada, Eastern Society of Anesthetists (first President).

Being of an inventive nature he was constantly constructing and improving anesthesia apparatus. He contributed appreciably to the safety and accuracy of these machines. He also completed a motion picture on the use of anesthetics.

By his death our staff has lost one of its most valued members. His advice on anesthesia problems was frequently sought and always cheerfully given. We shall miss his unusual talents, which were freely given to the hospital for many years. While extending our sympathy to his family, we wish to express our appreciation for the many years of faithful and unselfish service which he rendered.

P. F. Butler, *Secretary, Senior Staff.*

January 25, 1934.

IN MEMORIAM

DR. GEORGE L. RICHARDS

Dr. Richards was born in Farmington, Connecticut, June 1, 1863, and died in West Dennis, Massachusetts, November 9, 1933.

For several years he was on the Consulting Staff of the Cape Cod Hospital. His specialty was the ear, nose and throat, and in that branch of medicine and surgery he was a national figure. In 1930 he was elected to the Attending Staff.

By his passing it is but fitting that in tribute to his memory, we should speak of the excellencies of his good deeds.

He was a man of great civic pride and always took a prominent part in the affairs of the community. One could not fail to be impressed with his high Christian character and integrity. He also had that rare and mysterious quality which enabled him to see the best in everyone and it attracted to himself a legion of friends.

It was Dr. Richards' desire and ambition to make our Hospital not only a place of service to the community but a real haven for the comfort of those in distress of body and in peril of pain and death. He understood its problems as only a few men have and gave of himself unsparingly to its service. Those of us who knew him, served with him, and understood him, seemed to catch something of the contagion of his pluck and patience. We came to appreciate the warm current of underlying kindliness which ran through his whole life.

We shall feel his loss keenly at the Staff meetings and in the consultation room where his presence was always an inspiration and a welcome and dependable asset.

As a surgeon he was skilful, as a citizen he was a keen and understanding observer of world affairs, as a friend he was steadfast and true.

Be it resolved: that the heartfelt sympathy of the members of the Cape Cod Hospital Staff be extended to his family in their bereavement.

Be it further resolved: that the memorial be spread upon the records of the Cape Cod Hospital Staff, and a copy thereof be sent to the family of the deceased.

Dr. E. S. Osborne
Dr. J. P. Nickerson
Dr. C. E. Harris
Committee.

NOTICES

RADIO HEALTH MESSAGES

FEBRUARY-MARCH, 1934

Sponsorship: Public Education Committee of the Massachusetts Medical Society and Massachusetts Department of Public Health.

Courtesy WBZ. Fridays, 4:30 P.M.

February
9 Milk
16 Stomach Trouble
23 Lumps in the Neck

March
2 Age and Cancer
9 Some Problems of Epilepsy
16 Fractures
23 How to Keep the Well Child Well
30 Résumé of the Year's Work

HEALTH QUESTION BOX

Sponsored by Massachusetts Department of Public Health. Fridays 4:40 P.M.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 5:00 P.M.

Questions on Health and Prevention of Disease may be sent to: Radio Health Forum, State Department of Public Health, State House, Boston.

SPECIAL

From Friday, January 19, the State House Broadcasts were discontinued and in their place were substituted Ten Minute Health Reviews.

Sponsored by the Massachusetts Department of Public Health. Assisted by Miss Violette Babcock, Violinist, and Mr. G. Lambert Roscoe, Pianist and Organist.

Courtesy WEEI. Fridays, 1:15 P.M.

Glimpses into the History of Public Health in Massachusetts together with the Functions and Activities of the Massachusetts Department of Public Health, Blended with Classical Music.

After hearing this new program we would appreciate your comments.

REMOVAL

EDWARD R. WILLIAMS, M.D., announces the removal of his office to 320 Commonwealth Avenue, Boston.

REPORTS AND NOTICES OF MEETINGS

THE BOSTON TUBERCULOSIS ASSOCIATION

The annual meeting of the Boston Tuberculosis Association was held at 35 Tyler Street, Boston, January 30, 1934. The expenditures for the year were $41,532.02. The unexpended balance was $5.11. The Association's deficit is $3,412.87 to which the small balance will be applied.

The following officers were elected: President, Dr. John B. Hawes, 2d; Vice President, Dr. James J. Minot; Treasurer, George S. Mumford; and Mrs. Reginald Heber White, Clerk.

The executive committee consists of Dr. John B. Hawes, 2d, Chairman; Adolph Ehrlich, Dr. Cleaveland Floyd, Miss Sarah A. Hyams, Dr. Donald S. King, Henry C. McKenna, Dr. James J. Minot, Everett Morss, Jr., George S. Mumford, Dr. Joseph H. Pratt, Miss Julia C. Prendergast, Alexander Wheeler, Mrs. Reginald Heber White, Dr. Charles F. Wilinsky, and Dr. Nathaniel K. Wood.

AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE

SECTION N (MEDICAL)

WEDNESDAY MORNING SESSION, DECEMBER 27, 10:30 A.M.

John Ware Hall, Boston Medical Library

Dr. George Bigelow of the Massachusetts General Hospital presided in place of the Chairman, Dr. C. R. Stockard, who was ill.

The opening paper was presented by Dr. Reuben L. Kahn, developer of the well-known Kahn reaction, on "Tissue Reactions in Immunity." Dr. Kahn pointed out that present conceptions of immunity, other than those of a toxin-antitoxin nature, assume the primary importance of the body fluids, notably the blood, but do not account for those reactions observed in the skin and other tissues. The hypersensitive reactions of these in the sensitised animal, he believes, can be explained on a common basis with the reactions occurring in the blood, and the body tissues, of the immunized animal which have undergone one basic change, that of acquiring the ability to collect and react with antigen, thereby protecting the organism. In blood, phagocytosis occurs in fixed tissues, and localization of the toxic material takes place, preventing access to the blood and other fluids and hence to the body generally. Dr. Kahn bases this on work with albino rabbits in which he believes he has shown that the fixed tissues are not neutral bystanders while the body fluids react to toxins given intra- and subcutaneously, and intraperitoneally. Using horse serum intracutaneously as antigen, he obtained reactions from the tissues after precipitin and other blood responses had disappeared. Using horse serum antitoxin, by which means quantitative measures could be employed, he found that the number of units required to protect sensitized animals from additional injections of toxin was over 50 times the amount needed for non-sensitized controls receiving the same doses intracutaneously. This he explained as being due to the reaction of the fixed tissues to

horse serum, which localized the antitoxin in the sensitized animals with resultant non-absorption and failure to reach the toxin injected into the blood. Subcutaneous, intraperitoneal, intramuscular, and intracerebral injections of the serum-antitoxin mixture gave similar reactions, although less marked in the last two.

If the assumption of the primary importance of the fluid tissues were true, these reactions should have been alike. Dr. Kahn concluded that changes of all the tissues in immunity are unified processes in which the fixed tissues function by detecting, anchoring, and so preventing the spread of toxins with subsequent protection to the organism.

210
34

Dr. Irving J. Walker of the Boston City Hospital presented the second paper, his subject being "Why the Increasing Mortality in Appendicitis?" He believes that while more deaths are recorded as due to appendicitis, according to vital statistics, a misunderstanding or misinterpretation has arisen because the incidence of operations has not been taken into account. A study of the relation of deaths to the incidence of operations, past and present, should give a truer picture. Certain other factors, as changed and improved methods of diagnosis, ascribing pre and postoperative deaths due to complications as due to appendicitis, would lead to higher rates. He concludes, in part, that from a study of vital statistics the rate is rising; from the incidence of operations, the rate is falling; that there has been a misunderstanding due to the different methods employed in gathering figures and in not considering the change in incidence of operations. He warns against operations by the occasional operator and against operations for so-called chronic appendicitis.

The third paper was read by Dr. Herbert L. Lombard of the Massachusetts Department of Public Health, who spoke on "Results of an Appendicitis Campaign." He said that working somewhat along the lines of a campaign in this state against cancer, an attempt was made to educate the public to the danger of delay in seeking medical advice (arbitrarily defined as any period over 24 hours after the onset of symptoms) and use of laxatives for abdominal distress. The results of the campaign were not so marked as were those obtained in the work against cancer where a greater amount of attention was given to the use of newspaper advertisements. He believes that this agency was neglected or else the population at large is more apathetic to appendicitis.

Wednesday Afternoon Session, December 27

Symposium on Pneumonia

Dr. Lloyd D. Felton of the Harvard Medical School began the symposium with the subject "Pneumococcus Antibodies, What Are They?" He has found that the protective substance is little if at all organotropic, but highly bacteriotropic, is water-insoluble and difficult to study because of no sufficiently delicate protein test and the small amounts used. He found also that the antigen-antiserum union could be made reversible to about 85 per cent. He concludes that at present we cannot say the active antibody is anything but a protein-like compound, perhaps of a new type as its isoelectric point of 6.8-7.4 would indicate. Possibly this may be separated into active non-protein fractions, although this has as yet to be done.

W. F. Wells of the Harvard School of Public Health presented the next paper, discussing "Viability of Bacteria in Air." He assumes that organisms enter the air as the nuclei of droplets and depending on the size of these latter, the temperature, humidity, and viability of the bacteria, the ultimate deposition follows. In general the larger droplets go but a short distance before reaching the floor; as the size decreases, the interval before leaving the atmosphere increases and may be greater than the viability of the organisms. Using an experimental chamber, spores were obtained from the air after it had been quiet for a week. Pfeiffer's bacillus disappeared in 80 minutes; pneumococci, streptococci, and diphtheria organisms were cultured after one day; and staphylococci after three days. Sprays of the colon-dysentery group gave negative cultures after a few hours. An air centrifuge for obtaining cultures from known amounts of air and at the same time planting them on the desired media was shown.

Dr. Smillie, also of the Harvard School of Public Health, spoke on "The Epidemiology of Lobar Pneumonia—Recent Studies." Working on Types I and II, a series of contacts were studied; these were divided into family and hospital contacts for each source of contact. It was found that cultures of those exposed to Type I showed that 1.6 per cent of the hospital contacts became carriers of a homologous strain, while the rate was 24.2 per cent for the families. In Type II the rates were 3 per cent and 16.2 per cent, respectively. It was also noted that family contacts had as high a rate after an exposure of one day as after several. Colds made little difference, although 96 per cent of those acquiring homologous strains by contact had them. In summary, Dr. Smillie states: 264 contacts with Type I or II pneumonia were studied; 20 per cent of the family contacts and 2.3 per cent of those in hospitals acquired an homologous strain of one or the other of these organisms. Hence it is probably safe to allow patients with pneumonia on general wards, the number of those becoming carriers by contact who develop a corresponding pneumonia being very small.

Dr. Frederick T. Lord of the Massachusetts General Hospital presented the last paper, taking "Serum Treatment in Pneumonia" as his subject. Serum treatment is at present limited to cases of Types I and II, which account for approximately 57 per cent of all pneumonias. The ages between 28 and 50 years are the ones typically affected and postoperative cases, children, and vascular failures

are usually exempt from infection with these types.

Dr. Lord mentioned the use of the Sabin modification of the Neufeld method of typing—sputum containing the organisms is obtained and when these are brought into contact with rabbit antiserum of an homologous strain, marked swelling of the capsule occurs. The advantages of this method are that the typing can be done at the bedside and requires but a few minutes, with consequent saving of time in deciding on the use of antiserum.

With treatment in Type I cases in the first three or four days, 10.2 per cent of patients have died in a group studied in Massachusetts; and 24 per cent when untreated. In Type II, less complete figures are available but show approximately a 40 per cent mortality without serum and 29 per cent with it. Other types have not shown a response to antisera as yet.

There are certain difficulties in giving treatment—the patients are seen too late in general hospitals; laboratories are few; the antiserum expensive; general practitioners see relatively few cases. For the last three years a fund has been available in this state to study the treatment of lobar pneumonia, technicians and Felton's antisera being furnished. An extension of a year has made it possible to continue this for what will be a total of four years.

Dr. Lord concluded with a description of the dosages employed, restrictions and precautions to be observed in treating pneumonia patients with antisera.

The meeting was continued on Thursday with a Symposium on Sociology and Medicine in the morning session. The first speaker was Dr. Henry E. Sigerist of Johns Hopkins University, whose subject was "The Interrelationship of Medicine and General Culture." He stated that among the Semites sickness carried the odium of sin; among the Greeks, of inferiority; that later, notably during the Renaissance, the sense of odium was lost and suffering was believed good for the soul, the sick receiving a preferential place in society, as they now do. He believes that present changed conditions require a program for guidance and doubts if the Fascist system in Italy and Nazist in Germany will be productive.

Mr. Henry S. Dennison of Framingham, Mass., followed with "Medicine in Relation to Social Trends." He notes a turn toward organization and consolidation in medicine as in industry and government. He believes an increase of routine and preventive care among the poorer classes is needed and would open a new "market" for medicine without eliminating the individual doctor in the same way that the chain store has not ended the local store. He thinks there is a great need for the doctor's knowledge of human nature in politics and economics and hopes that there will be a fulfillment of this.

The third paper was by Douglass V. Brown of the Harvard Medical School who spoke on "Some Economic Aspects of Medicine." He did not attempt the solution problem of the individual doctor; he believes much progress might well be made in medicine in future legislation, the technical aspect not being the only source of advancement.

"Teaching the Social Implications of Sickness to Medical Students" was given by Ida M. Cannon of the Massachusetts General Hospital, Boston. She stressed the importance of "social" case teaching—the need of drawing attention to the social, temperamental, and environmental aspects as well as the medical side of the patient for a complete understanding.

Dr. George R. Minot, Boston City Hospital, concluded the symposium with "Social Medical Practice." He feels that the diagnosis of the person is too often neglected for the diagnosis of the disease. At present, the doctor's acquaintance with the patient other than as a case of a certain disease requires a conscious effort and is not automatic as it was 100 years ago; this is especially true in hospitals, where social service is a needed and important organization. The private doctor, while calling in specialists, must remember to look out for this side of his patient. Long sickness as in peptic ulcer and arthritis especially need such attention, an adjustment of the views of life may even be necessary. The results well repay the effort made.

Thursday afternoon was devoted to a symposium on Tuberculosis. Dr. William C. White of the National Tuberculosis Association, Washington, D. C., talking on "The Biology of the Tubercle Bacillus," opened the meeting. He discussed the life cycle apart from multiplication by binary division and collection of monocytic cells about a primary focus of infection, which the acid-fast organisms of leprosy and Johné's disease of cattle also call forth. He described the method of large scale culturing on artificial media as that devised by Long, by which a study of the chemistry of the organism and its products as well as the effect of changes of the constituents could be effected. Direct observation, aided by photography, and a study of the metabolism of the bacillus are also being used. We are still ignorant, however, of what goes on when the organism is outside the body.

It has been found that all types are of a virulent or rough, a less virulent or smooth type and, in the case of the avian type, the change by cultural methods from rough to smooth has actually been carried out.

All the acid-fast group have common compounds, but the human type has been found to have a peculiar fatty acid which in pure form produces all the reactions of the tubercle bacillus in the body from the attraction of monocytic cells to caseation, without the presence of live organisms. The skin reactions are produced by a protein compound peculiar to human and bovine strains. At present the old tuberculin used in skin testing varies as much as

400 per cent and it is hoped that a standard will be developed to do away with this variation. Other chemical compounds have not as yet been found which have any individualism in respect to their occurrence in certain strains only.

Finally, no specific treatment has been devised for infection of the human host by the tubercle bacillus.

Dr. Henry D. Chadwick of the Massachusetts Department of Public Health was the second speaker, presenting "The Incidence of Tuberculosis" with special reference to Massachusetts. In this state the period of greatest freedom is in those under the age of ten years. After this there is a more or less steady rise to the age of 70 or 75 in males; females show a somewhat sharper rise with a smaller total to the 20-29 year group, a slow decline to 55 years, and a final increase nearly reaching the level of the other sex. More females are affected in early middle life, but more males eventually die. It should be noted that the popular idea that once past 45, the dangers from tuberculosis are over is erroneous, as it actually is the reverse.

Dr. Edward D. Churchill of the Massachusetts General Hospital presented the last paper "Surgery in Tuberculosis." He believes there are no definite rules for the indications for surgery, the physician and surgeon together being needed to decide. However, operation is not a last resort as it once was, present results being comparable to those obtained in peptic ulcer operations for example. He states that surgery acts by immobilizing the affected lung, allowing healing fibrosis; obliterates cavities; reduces the amount of retained sputum; prevents or arrests hemorrhages and retards the progress of the disease. The type of operation has changed somewhat — Sauerbruch's thoracoplasty has been abandoned as not giving sufficient collapse, a wider resection of ribs with removal (complete) of the first two in apex infections being advocated. The use of more than one-stage operations has likewise been of aid. A partial collapse of one apex with a functioning lower lobe can be done with success. Phrenic nerve paralysis is attended with no permanent loss of function, as is phrenicotomy, and should be remembered in questionable cases. Cavitation should not be treated by these measures.

The importance of proof of the clinical diagnosis is great, as other lung conditions are now being operated on and it may make a great difference to the patient, as more and more favorable results are following operations for cancer and bronchiectasis with surgical treatment. A loose diagnosis of tuberculosis may be a dangerous thing. Dr. Churchill believes large hospitals or any institution handling tuberculosis should have a prediagnosis ward where all patients are kept until positively diagnosed by culture, smear, or other means; until this has been done, treatment cannot properly be decided on.

On Friday, December 29, 1933, the morning session was devoted to a joint meeting of the American Pharmaceutical Association with the American College of Dentists. Dr. Theodore Bradley presided. In the afternoon and evening of the same day, there was a joint meeting of the American College of Dentists with Section N. Several short papers on problems of dental and medical interest were presented.

On Saturday morning, December 30, 1933, the American Society of Parasitologists met with Section N. Short papers were presented by Drs. Hans Zinsser, Henry Pinkerton, S. B. Wolbach, and Marshall Hertig of the Harvard Medical School, and by Dr. R. R. Parker of the United States Public Health Service.

THE WORCESTER NORTH DISTRICT MEDICAL SOCIETY

The presence of President William H. Robey of the Massachusetts Medical Society accounted for the most largely attended meeting of recent years of the Worcester North District Medical Society at the Leominster Hospital, Wednesday, January 24. Dr. Robey spoke on matters pertaining to the work of the Federal Emergency Relief and the C.W.A. He was able to clear up many uncertainties in the minds of the members in relation to these two programs. His talk was very enlightening.

Dr. Thomas J. O'Brien was also present and related matters of interest in regard to legislative bills that are now pending. Both speakers brought to the members an enthusiasm that was greatly appreciated.

About 60 doctors enjoyed a splendid turkey dinner served by the wives of the Leominster members of the Society.

FRANCIS M. MCMURRAY, M.D., *Secretary.*

THE MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

The Massachusetts Association of Boards of Health, one of the oldest public health organizations in this country, at its annual meeting at The University Club, Boston, Thursday, January 25, declared for the formation of regional chapters of specialized public health workers.

Only those who are members of the association are eligible to membership in the regional chapters which will elect their own officers and have representation on the Executive Committee of the association. The regional chapters will hold meetings independent of the association but the annual meeting of each chapter will be held on the same day and at the same place as that of the State Association.

The Executive Committee in the past has been composed of five elective members and the past presidents ex-officiis, and the President has appointed the committee on nomination of officers. A change was made in this latter procedure so that hereafter a nominating committee of three will be elected at the October meeting, each member eventually to serve for five years and each not to be eligible to reëlection.

Dr. Francis P. Denny of Brookline, Dr. Geo. Francis

Curtis of Newton, Dr. George H. Bigelow, former State Health Commissioner, and Prof. Curtis M. Hilliard of Simmons College, each of whom served on the committee recommending the changes in the constitution, were delegated to proceed with the reorganization of the association as planned.

State Health Commissioner Henry D. Chadwick explained bills affecting public health now before the Legislature.

The officers elected for the current year (the association to function as in the past until the regional chapters are formed) are the following:

President, Joseph B. Glennon, New Bedford.

First Vice-President, Dr. Paul N. Withington, Milton.

Second Vice-President, Dr. Ernest M. Morris, Fall River.

Secretary-Treasurer, George T. Lennon, Haverhill.

Elective members of the Executive Committee: Dr. M. Victor Safford, Boston; Dr. William O. Hewitt, Attleboro; Dr. Peter O. Shea, Worcester; Dr. J. H. Lawrence, Brockton; Dr. Curtis M. Hilliard, Wellesley.

THIRTIETH ANNUAL CONGRESS ON MEDICAL EDUCATION, LICENSURE AND HOSPITALS

FEBRUARY 12 AND 13, 1934

Palmer House, Chicago

PROGRAM

MONDAY MORNING, 10:00

Review of the Accomplishments of the Council on Medical Education and Hospitals. Ray Lyman Wilbur, M.D., Chairman, Stanford University, California.

Philosophy of Professional Licensure. Justin Miller, J.D., Dean, Duke University School of Law, Durham, N. C.

Discussion: Alphonse M. Schwitalla, S.J., Ph.D., St. Louis.

Medical Education and Its Relationship to Society as a Whole. Robert G. Sproul, LL.D., President, University of California, Berkeley.

Discussion: E. P. Lyon, M.D., Minneapolis.

The Restoration of the General Practitioner. Dean Lewis, M.D., President, American Medical Association, Baltimore.

Discussion: James B. Herrick, M.D., Chicago; J. H. Musser, M.D., New Orleans.

Red Lacquer Room

MONDAY AFTERNOON, 2:00

Joint Session of the Council on Medical Education and Hospitals and the American Conference on Hospital Service

Merritte W. Ireland, M.D., Presiding

Responsibility of the Hospital Trustee and the Relationship Between the Trustees and the Staff. Howard S. Cullman, President, Beekman Street Hospital, New York.

Discussion: Nathaniel W. Faxon, M.D., Rochester, New York.

Symposium: Size and Scope of a University Clinic.
Henry Houghton, M.D., Director, University of Chicago Clinics.
Nathan B. Van Etten, M.D., Vice Speaker, House of Delegates, American Medical Association, New York.
John H. J. Upham, M.D., Dean, Ohio State University College of Medicine, Columbus.

Discussion: William D. Haggard, M.D., Nashville; John Wyckoff, M.D., New York; Austin A. Hayden, M.D., Chicago.

Red Lacquer Room

TUESDAY MORNING, 9:00

Joint Session of the Council on Medical Education and Hospitals and the Federation of State Medical Boards of the United States

G. M. Williamson, M.D., Presiding

The Privilege of Reëxamination in Professional Licensure. Bernard C. Gavit, J.D., Dean, Indiana University School of Law, Bloomington.

Discussion: G. M. Williamson, M.D., Grand Forks, N. D.

Résumé of the History and Present Application of Medical Licensure in the States. J. N. Baker, M.D., Secretary, Alabama Board of Medical Examiners, Montgomery.

Discussion: A. T. McCormack, M.D., Louisville.

Reciprocity Agreements. J. R. Neal, M.D., Secretary, Professional Committee for Medicine, Illinois Department of Registration and Education, Springfield.

Discussion: Harold Rypins, M.D., Albany, N. Y.

Medical Licensure in South America—Preliminary Survey. William D. Cutter, M.D., Secretary, Council on Medical Education and Hospitals, Chicago.

Discussion: Frederic W. Schlutz, M.D., Chicago.

Red Lacquer Room

TUESDAY AFTERNOON, 2:00

Reginald Fitz, M.D., Presiding

The Importance of Introducing Psychiatry Into the General Internship. Franklin G. Ebaugh, M.D., Director, Division of Psychiatric Education, The National Committee for Mental Hygiene, Denver.

Discussion: C. C. Burlingame, M.D., Hartford, Conn.

The Incorporation of the Principles of Preventive Medicine in Clinical Teaching. Wilson G. Smillie, M.D., Professor of Public Health Administration, Harvard University, Boston.

Discussion: C. Sidney Burwell, M.D., Nashville.

The Teaching of Industrial Hygiene. Leverett D. Bristol, M.D., Health Director, American Telephone and Telegraph Company, New York.

Discussion: Edward C. Holmblad, M.D., Chicago.

The Function of the Physician in Public Health Education. W. W. Bauer, M.D., Director, Bureau of Health and Public Instruction, American Medical Association, Chicago.

Discussion: H. S. Cumming, M.D., Washington, D.C.

Red Lacquer Room.

TUESDAY AFTERNOON, 2:00

The Federation of State Medical Boards of The United States

G. M. Williamson, M.D., Presiding

Traffic in Narcotics by Licensed Physicians. H. J. Anslinger, United States Commissioner of Narcotics, Washington, D.C.

Discussion: W. L. Treadway, M.D., Washington, D.C.

The Use of the Injunction Procedure in Enforcing Medical Practice Acts. F. Manley Brist, LL.B., Attorney, Minnesota Board of Medical Examiners, St. Paul.

Discussion: H. M. Platter, M.D., Columbus, Ohio.

Room 14

Federation Dinner. Monday, 6:30, Palmer House. The Federation of State Medical Boards of the United States. Address: Relation of Education to Licensure. Walter A. Jessup, Ph.D., President, State University of Iowa, Iowa City. Presidential Address by G. M. Williamson, M.D. Informal round table discussion.

Evening Meeting. Monday, 8:15, Red Lacquer Room, Palmer House. American Conference on Hospital Service. Address: The Old and the New in Medicine. Dean Lewis, M.D., President, American Medical Association, Baltimore.

Luncheon. Tuesday, 12:30, Grand Ball Room, Palmer House. Central Council for Nursing Education. Address: What Is the Future of Nursing. Hugh Cabot, M.D., Professor of Surgery, University of Minnesota Graduate School of Medicine, Minneapolis.

REDUCED RAILWAY FARES

Reduced railway fares will be in effect for those who attend the Congress. In some cases excursion rates are offered. In most instances, however, the reduced rate will be upon the certificate plan and it is necessary to obtain a *certificate* at the time of purchasing. After the certificate has been countersigned by a representative of the railroads at the Congress, it will entitle the holder to a reduction on the return trip.

PHI DELTA EPSILON FRATERNITY, BOSTON UNIVERSITY SCHOOL OF MEDICINE

There will be an open meeting of the Phi Delta Epsilon Fraternity of the Boston University School of Medicine at the Evans Memorial Hospital Auditorium, 80 East Concord Street, Boston, on Friday, February 16, 1934, at 8 P.M.

The topic for discussion will be: The Treatment of Angina Pectoris and Congestive Heart Disease by the Total Ablation of the Normal Thyroid Gland.

The speakers:

Medical aspect: Dr. Herrman L. Blumgart.

Surgical aspect: Dr. David Berlin.

The discussion will be led by Dr. William D. Reid. Physicians and medical students are invited.

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, February 20, 1934, at 12 noon. The speaker will be William C. Quinby, M.D., Clinical Professor of Genito-Urinary Surgery, Harvard Medical School; Urologist, Peter Bent Brigham Hospital. His subject will be "Tumors of the Genito-Urinary Organs," this to be illustrated with Lantern Slides. All physicians are cordially invited to attend. The usual luncheon will be served at 1 o'clock.

MASSACHUSETTS ITALIAN MEDICAL SOCIETY

The regular monthly meeting of the Massachusetts Italian Medical Society will be held in the Hotel Westminster, Boston, February 12, at 8 P.M. Telephone Kenmore 5100.

Business.

Communications.

"Obstetrical Conservatism with a Résumé of 1000 Cases." Dr. B. Macchia.

General Discussion.

The medical profession and medical students are cordially invited.

CARL F. MARALDI, M.D., *Secretary.*
276 Commonwealth Avenue, Boston.

MASSACHUSETTS MEMORIAL HOSPITALS

The monthly meeting of the Surgical Section will be held in the Trustees Room of the Hospital on Friday, February 9, 1934, at 12 noon.

Dr. W. D. Rowland will address the members on "Common Problems of the General Surgeon and Ophthalmologist."

RALPH C. WIGGIN, *Secretary.*

NEW ENGLAND WOMEN'S MEDICAL SOCIETY

The fifty-sixth annual meeting of the New England Women's Medical Society will be held at The Hotel Vendome, on Thursday, February 15, 1934. Business meeting at 6:30. Dinner at 7 o'clock. Speakers will be Mrs. Eva Whiting White and Miss Sybil Holmes.

ELIZABETH L. BROYLES, *Secretary.*

THE MASSACHUSETTS PHYSIOTHERAPY ASSOCIATION

The Massachusetts Physiotherapy Association will meet at the Hotel Kenmore on February 13 at 8:00 P.M. Dr. Richard M. Smith will speak on the subject "The Cost of Medical Care."

BOSTON UNIVERSITY SCHOOL OF MEDICINE

WILL CONDUCT A CLINICAL MEETING AT BOSTON CITY HOSPITAL

Dr. E. Starr Judd, Professor of Surgery, University of Minnesota School of Medicine and Surgeon at the Mayo Clinic, Rochester, Minnesota, will give a clinic in the Cheever Amphitheatre, Boston City Hospital, at 11 o'clock on Monday, April 16, under the auspices of the Department of Surgery of Boston University School of Medicine.

His subject will be "Surgical Diseases of the Biliary Tract." Drs. Horace Binney of the Boston City Hospital, Arthur W. Allen of the Massachusetts General Hospital and Elliott C. Cutler of the Peter Bent Brigham Hospital will take part in the discussion.

Physicians and medical students are invited to attend.

OTTO J. HERMANN,
WILLIAM R. MORRISON,
DWIGHT O'HARA,
Committee on Clinics,
Boston City Hospital.

THE MALDEN MEDICAL SOCIETY

The next meeting will be held on February 20, at Malden Electric Co. Hall, at 8:30 P.M.

Speaker: Dean Warren Stearns.

Subject: Outline of Psychiatry.

A. H. WARREN, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance), Tuesday evening, February 13, at 8:15 o'clock.

PROGRAM

Presentation of Cases. Dr. C. F. McKhann.

The Mechanism of Salt Deficit in Chronic Nephritis. By Dr. A. M. Butler.

The Specific Treatment of Influenzal Meningitis. By Dr. L. D. Fothergill.

Some Experiments in Iron Metabolism. By Dr. L. K. Diamond.

JOHN HOMANS, M.D., *Secretary.*

MASSACHUSETTS GENERAL HOSPITAL

A Clinical Meeting of the Staff of the Massachusetts General Hospital will be held in the Moseley Memorial Building, on Thursday, February 15, 1934, at 8:15 P.M.

PROGRAM

1. Diagnosis of the Hemorrhagic Tendency in Obstructive Jaundice. Dr. R. R. Linton.
2. Medical Significance of Gall Bladder Disease. Dr. C. M. Jones.
3. Surgical Aspect of Cholelithiasis and Its Complications. Dr. L. S. McKittrick.

Physicians, medical students, nurses and social workers are cordially invited.

Committee on Hospital Meetings.

THE NEW ENGLAND ROENTGEN RAY SOCIETY

The February meeting will be held at the Boston Medical Library, Friday night, February 16, 1934.

SCIENTIFIC SESSION

"Radiography as Aid to Renal-Tract Diagnosis."
Bryant D. Wetherell, M.D., Boston, Mass.

T. R. HEALY, M.D., *Secretary.*
370 Marlborough Street,
Boston, Mass.

NEW ENGLAND HEART ASSOCIATION

The fifth meeting of the New England Heart Association will be held in the Amphitheatre of the Children's Hospital, Monday, February 26, at 8:15 P.M.

All members of the New England Heart Association and interested physicians are invited to attend.

PAUL W. EMERSON, M.D., *Secretary.*

NEW ENGLAND SECTION OF THE ILLUMINATING ENGINEERING SOCIETY

There will be a meeting of this Society on Friday, February 16, at 7:45 P.M. in the Amphitheatre of Building C of the Harvard Medical School.

Program: Sunlight and Artificial Sunlight in Health and Disease, by Dr. E. T. Wyman. Industrial and Medical Applications of Ultraviolet, by Dr. M. J. Dorcas.

Members of the medical profession are cordially invited.

D. P. STONE, *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

February 9—Massachusetts Memorial Hospitals. See page 341.

February 9—William Harvey Society at the Beth Israel Hospital, at 8 P.M. Speaker: Dr. George Blumer, Yale Medical School. Subject: "Bedside Diagnosis."

February 12—House Officers' Association of the Boston City Hospital. 8 P.M. Subject: Endocrinology. Speakers: Drs. Albright, Rowe, Cochrane, Aub.

February 12—Massachusetts Italian Medical Society. See page 341.

February 12, 13—Thirtieth Annual Congress on Medical Education, Licensure and Hospitals. See page 340.

COURSE ON MEDICAL BIBLIOGRAPHY

Boston Medical Library

February 13—Incunabula. James F. Ballard.
Tuesday at 8 P.M.
Boston Medical Library, 8 The Fenway.

February 13—Massachusetts Physiotherapy Association. See page 342.

February 13—Harvard Medical Society. See page 342.

February 14—New England Dermatological Society will meet at 3 P.M. at the Massachusetts General Hospital.

February 14—Greater Boston Medical Society will meet at the Beth Israel Hospital, Boston, from 8 A.M. to 4:30 P.M.

February 15—Massachusetts General Hospital, Clinical Meeting of Staff. See page 342.

February 15—New England Women's Medical Society. See page 341.

February 16—Phi Delta Epsilon Fraternity. See page 341.

February 16—New England Section of the Illuminating Engineering Society. See page 342.

February 16—The New England Roentgen Ray Society. See page 342.

February 16 and 17—The New England Hospital Association is holding its Twelfth Annual Meeting at the University Club, Boston. For details write Dr. A. G. Engelbach, Massachusetts General Hospital, Boston.

February 20—South End Medical Club. See page 341.

February 20—Malden Medical Society. See page 342.

February 26—New England Heart Association. See page 342.

March 5, 6 and 7—The Southeastern Surgical Congress will be held at Nashville, Tenn. For information write Dr. B. T. Beasley, 1019 Doctors Building, Atlanta.

March 9—William Harvey Society at Beth Israel Hospital. Speaker: Dr. Irving J. Walker, Clinical Professor of Surgery, Harvard Medical School. Subject: "Judgment and Conscience in Surgery."

March 12—House Officers' Association, Boston City Hospital, 8:00 P.M. Speakers: Drs. A. Warren Stearns, Abraham Myerson. Subject: Forensic Psychiatry.

April 16—Boston University School of Medicine to Conduct a Clinical Meeting at Boston City Hospital. See page 342.

April 16-20—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

April 30—The American Board of Dermatology and Syphilology. Examinations for Certificates. Address: Dr. C. Guy Lane, 416 Marlboro Street, Boston, for details.

July 24-31—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schnis. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

September 3-6—American Public Health Association, at Pasadena, California. Dr. J. D. Dunshee, Chairman, Local Committee on Arrangements.

September 4, 5, 6—International Union Against Tuberculosis will be held in Warsaw. For particulars address The National Tuberculosis Association, 450 Seventh Avenue, New York, N. Y.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Wednesday, March 7—Lynn Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Frank H. Lahey, Boston. Subject to be announced. Film Electrocardiogram.

Wednesday, April 4—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speakers: Dr. Elliott P. Joslin and Dr. Howard F. Root, Boston. Subject: "Tuberculosis Complicating Diabetes."

Thursday, May 3—Censors' Meeting, at Salem Hospital, 3:30 P.M.

Tuesday, May 8—Annual Meeting. Salem Country Club, Forrest Street, Peabody. Dinner at 7. Speaker to be announced. Subject to be announced.

RALPH E. STONE, M.D., Secretary.
221 Cabot Street, Beverly, Mass.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland, Mass.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Meetings will take place in March (2nd Wednesday) at Wakefield, and May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, M.D., Secretary.
76 Church Street, Winchester, Mass.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meeting will be held on April 25.

T. A. STAMAS, M.D., Secretary.
226 Central Street, Lowell, Mass.

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

February 20—Meeting at the Metropolitan State Hospital, Waltham, 5 P.M.

NORFOLK DISTRICT MEDICAL SOCIETY

February 27—Hotel Kenmore, 8:30 P.M. Dr. J. H. Shortell: "Industrial Medicine and Surgery."

March 27—Faulkner Hospital, 8:30 P.M. Dr. Henry H. Faxon and Dr. Edward A. Edwards: Symposium on "Varicose Veins." Discussion by Dr. E. E. O'Neil.

April 17—Hotel Kenmore, 8:30 P.M. Special Business Meeting.

May—Annual Meeting. Time, place and program to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline, Mass.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

March 1—12 noon at Quincy City Hospital. Program by the hospital staff.

April 5—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.

May 3—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

N. R. PILLSBURY, M.D., Secretary.
Norfolk County Hospital, South Braintree, Mass.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 28—Clinical Meeting at the Massachusetts Memorial Hospitals.

April 25—Annual Meeting at the Boston Medical Library. Election of Officers. Scientific Program, titles and speakers to be announced.

The Medical Profession is cordially invited to attend all of these meetings.

JAMES H. MEANS, M.D., Vice-President.
GEORGE P. REYNOLDS, M.D., Secretary,
311 Beacon Street, Boston, Mass.

WORCESTER DISTRICT MEDICAL SOCIETY

All meetings to be held on Wednesdays as follows:

February 14—Dinner and scientific program at the Worcester State Hospital, Worcester, Mass.

March 14—Dinner and scientific program at the Memorial Hospital, Worcester, Mass.

April 11—Open date.

May 9—Annual Meeting. Time and place to be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester, Mass.

BOOK REVIEWS

Diet and Personality. Fitting Food to Type and Environment. By L. JEAN BOGERT. Published by The Macmillan Company. 223 Pages. Price $2.00.

The approval of Lafayette B. Mendel puts the dietetic principles and their practical application as found in this volume beyond possible criticism. In "Diet and Personality" the author has mixed sound fact with well-reasoned common sense and an adequate dash of humor to produce a book which even the most cautious physician may well recommend to his patients. What higher praise?

Obstetrical Nursing. By CAROLYN CONANT VAN BLARCOM. Third Edition. Published by The Macmillan Company. 651 Pages. Price $3.00.

The interesting and unique treatment of obstetrical nursing merits this third revision. It has been read with genuine enjoyment. The author has sincerely

emphasized the necessity for the simplification of nursing care both in the home and the hospital. The greater appreciation of the social implications of obstetrics is constantly urged upon the pupil. New developments in nutrition; roentgen pelvimetry; the physiology of gestation and the early diagnosis of pregnancy are fully discussed. The author develops her nursing creed in a unique introductory chapter on the "Meaning of Obstetric Nursing." The arrangement of the book, with many routine procedures in outline form makes it a work for easy consultation, and the rich supply of definitions and adequate illustrations should lead every teacher of nursing to endorse it for her pupils.

Pediatrics. By HENRY DWIGHT CHAPIN, M.D., and LAURENCE T. ROYSTER, M.D. Seventh Edition. Published by William Wood & Company. 775 Pages. Price $7.00.

This edition contains 750 pages dealing with many phases and diseases of infancy and childhood. It is almost encyclopedic, but like most encyclopedias it passes over some points in a superficial, if not evasive manner. Had the authors confined themselves to a smaller variety of subjects their book would be more interesting and probably more accurate.

The chapter on tuberculosis is out of date in some respects. Pulmonary infection is said to originate in "the lymph glands at the bifurcation of the trachea." Although a page of words is devoted to prophylaxis there is nowhere an account of modern case-finding methods.

The discussion of exanthemata is largely academic. Neither tuberculosis nor arteriosclerosis is mentioned as a liability for the diabetic child. One must be hypercritical, however, to hold such omissions against what is essentially a good text for students, and reference for physicians. The reviewer feels, nevertheless, that approximately one-third of the material could be advantageously deleted.

Colds and Hay Fever. By FRANK COKE. Published by William Wood & Company. 148 Pages. Price $2.00.

This is an attractive little volume written in a light conversational way. It would appeal to any intelligent layman and makes good reading for the doctor.

Dr. Coke is an Englishman who has already written a book on Asthma and knows his subject. Like all of us, he has his hobbies which he rides fast only to find that he must go back for details, and very often, these are given, sometimes more, sometimes less space than they deserve.

The normal functions of the nose are well described and then six pages are devoted to the physiology of the act of sneezing. The common cold is treated in its economic and hygienic aspects but Dochez' brilliant work with the virus deserves much more space. In hay fever and the allergic cause of sneezing, the author is evidently not quite convinced that allergy can explain the whole clinical picture. "Ionization," cautery and sprays must be used in addition. We know, however, that the great interest in allergy developed in America is not so highly developed on the other side. Dr. Coke is interested in the French conception of a disturbance of the colloidal particles in the blood and believes "paroxysmal rhinitis" may have such a cause. He advises intravenous doses of "collosal manganese" as well as ionization of the nose by electricity.

As a whole, the book is worth while. Another time, however, the arrangement could be improved and the space divided to better advantage.

Food Products. By HENRY C. SHERMAN. Third Edition. Published by The Macmillan Company. 674 Pages. Price $3.00.

A third edition of Dr. Sherman's book on food products is a valuable addition to the literature on nutrition. The subject is presented in an orderly and readily accessible form, for most of the text describes special foods, as milk and milk products, eggs, fish and game, meats, the products of grain, vegetables, nuts, fruits, etc. In these chapters, the production and marketing, the standards of purity according to the sanitary laws, and the composition, mineral and vitamin content, the digestibility, nutritive value and use of these foods are described. In other chapters, the general aspects of nutrition, the constituents, functions and control of food, and food economics are explained. In the appendix, the regulations of the Food and Drugs Act, as well as those of meat inspection are given. An unusual and valuable addition to each chapter is a list of books and articles for further study or better comprehension of the subject. A few references are also given. The text is illustrated by some good pictures and is clarified by many tables, the style of writing is easy, yet accurate, and the type is good. The book should be especially valuable for dietitians, and also for physicians and lay workers in nutrition.

Behind the Doctor. By LOGAN CLENDENING. New York: Alfred A. Knopf, 1933. xxi + 458 + xi pages. Price $3.75.

Of the popular books on medical history published in the last few years the book under consideration seems to the reviewer to be one of the best. The subject has been taken up in a lively and entertaining manner. Dr. Clendening has dramatized some of the important events in medical history, using as a background the actual contemporary publications. By this method, basing his work on sound conclusions, he presents a picture of historical events in a successful manner. The book is full of interesting material and splendid illustrations. It ought to have a wide appeal to the general public as well as to the medical profession. One or two minor errors were noticed, but these are easily offset by the general tone of the whole publication.